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THE BLOOD SUPPLY
TO THE HEART

THE BLOOD SUPPLY TO THE HEART

IN ITS ANATOMICAL AND CLINICAL ASPECTS

BY

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WITH AN INTRODUCTION BY HORST OERTEL

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WITH TWENTY-NINE FULL PAGE PLATES
AND SIX TEXT ILLUSTRATIONS



PAUL B. HOEBER
NEW YORK

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By PAUL B. HOEBER

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TO MY ALMA MATER
AND
ALL THOSE FROM WHOM SHE DREW
HER GREATNESS AND NOBILITY
THIS BOOK IS DEDICATED
ON THE OCCASION OF
THE MC GILL UNIVERSITY CENTENNIAL
OCTOBER, NINETEEN HUNDRED AND TWENTY-ONE

INTRODUCTION

THIS monograph on the blood supply to the heart in its anatomical and clinical aspects is the outcome of investigations which Dr. Gross commenced several years ago in the laboratories of the Royal Victoria Hospital and of McGill University. They formed originally part of a general study dealing with structural evolution of organs in the various age periods in its relation to normal function and disease.¹

It soon became apparent to me that in these researches Dr. Gross had, with rare industry and ingenuity, gone far beyond the original questions and that the results of his work touch upon a larger number of problems which are not only of anatomical, but also of great clinical interest and importance. The extent of the work and the accumulated material and illustrations had also gone far beyond the original scope of a paper and it was therefore considered advisable to publish it in monograph form. This allowed a further extension and inclusion of a thorough critical review, and incorporation of previous literature—by itself a useful undertaking. The work, therefore, places before us:

First: A complete description of the arterial and venous blood supply to the normal heart with a statistical study of its variations.

Second: The blood supply to the neuromuscular system of His and its pathological and clinical significance.

¹ See Oertel, Post-Natal Development and Pathological Organ Reconstruction in Relation to Function and Disease, *Am. J. M. Sc.*, May, 1921, p. 694.

Third: A new standpoint in the discussion of the anatomical factors concerning the etiology and development of valvular endocarditis.

Fourth: A newer view on the physiological course and character of the cardiac circulation in the various age periods and their relation to physiological and pathological functions.

The monograph contains, so far as I know, a complete presentation of the subject to date and possesses for anatomist, physiologist, pathologist and clinician alike an unusual combination of interest and usefulness.

Mr. Hoeber's rare good will to publish the work is an illustration of his unselfish, scientific spirit.

HORST OERTEL.

McGill University and The Royal Victoria
Hospital, Laboratories of Pathology.
Montreal, *July*, 1921.

PREFACE

THE author was primarily interested in the study of the circulation of the heart in its relation to age periods and in its pathological variations. For a full appreciation of the changes in the cardiac circulatory architecture under these conditions, it was found advisable to make a thorough study of the normal.

A review of the literature proved inadequate to allow an intelligent grasp of the subject and only served to perplex by its chaotic state. With much difficulty it was attempted to coordinate the various views, and there remained many points of dispute and a large number of problems. The author consequently made a personal study of the circulatory structure in the normal heart in order to investigate as many of the moot points as possible and also to acquire a standard for comparison with the results of future investigations into pathological conditions.

In the course of this research a technique was developed, which, it is believed, is eminently suited for investigations of this nature. Many of the disputed points have been, it is hoped, settled and a number of new and interesting fields for speculation, opened. Chief among the latter is the question of the effect of age on the course, character and physiology of the cardiac circulation. It was found that a very characteristic series of changes takes place as age progresses. This appears, by itself, an interesting contribution to the clinical

appreciation of the physiology and pathology of senile heart and death.

The blood supply to the neuromuscular system was worked out with care. It was found that the anatomy of the neuromuscular circulatory structure could be intelligently correlated with functional derangements such as presented by classical cases found in the literature.

Moreover, much of the reported experimental work acquired a new meaning. With the results of a series of experiments which the author himself undertook, the anatomical factors which enter into the production of valvular endocarditis have been put in a position which appears to throw new light upon the clinical and anatomical experiences of previous investigators.

In view of the accumulation of these facts and the work that was required to make a thorough search into the literature, Professor Horst Oertel suggested to present the whole matter in monograph form. It has consequently been decided to present in these pages a summary of the state of our knowledge on this subject.

For convenience in handling the matter, it has been divided into eight chapters. It is true that the divisions overlap somewhat, but in this way the historical review of the various problems is much more comprehensively handled.

The general plan is to give a concise historical survey of the matter dealt with in a chapter, indicating briefly the technique employed by the various investigators; then to outline the present state of knowledge and finally to add the author's contribution.

Appended is a bibliography arranged in alphabetical form. As far as possible, this was made complete. Unavoidably,

some contributions must have been left out, but it is hoped that the bibliography forms in itself a complete chain of evidence, reflecting the phases of thought through which the subject has passed.

The cross index should render the book of more ready reference and increase its usefulness.

The author wishes to take this opportunity to thank Prof. Horst Oertel for his generous and sympathetic advice throughout the work. To Dr. J. D. Morgan, roentgenologist of the Royal Victoria Hospital, and to Mr. Black of the United Photographic Store, Montreal, he is much indebted for the beautiful roentgenograms, plates and photographs which have made the work possible. To Mr. H. E. Webster, Superintendent of the Royal Victoria Hospital, who has kindly supplied apparatus and laboratory facilities, to Drs. Semple, Pitts, Waugh and Branch for autopsy material, and to his wife for invaluable aid in preparing the bibliography, the author also wishes to express his appreciation. Not the least of these, he owes thanks to Mr. Paul B. Hoeber for the kindly interest he has taken in publishing this work.

L. G.

McGill University and The Royal Victoria
Hospital, Laboratories of Pathology.
Montreal, *July*, 1921.

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CHAPTER I

TECHNIQUE

A. OLDER METHODS

THE earliest observations which are recorded on the blood supply to the heart, date from the middle of the sixteenth century. These were made purely on the basis of dissections. A number of these from Vesalius, Fallopius, Riolanus and others are found to be just as true to-day as at the time they were made.

Haller, whose detailed account of the coronary artery circulation was published in 1757, has given us a most remarkable illustration of what excellent results can be obtained by careful dissection.

Obviously, this method had its limitations; consequently, later investigators employed injections of the blood-vessels with metals, followed by corrosion of the tissues. Thus, Hyrtl in 1855 contributed a great deal to our knowledge of the more minute circulation in the heart as well as in other organs. His method consisted in injecting a metallic alloy of low melting point into the blood-vessels and subsequently placing the organ in a strong acid. After corrosion of the tissue, the metal cast was carefully washed free from debris. This method has been repeatedly employed by many investigators since Hyrtl and has been brought to a state of perfection by Nussbaum, whose contribution to our knowledge of coronary artery circulation in 1911, showed how such a bulky and awkward

mass as metal may nevertheless be used with very successful results.

Metal injections, however, were far from the ideal, since they could not be forced into the more minute channels. Moreover, the heaviness of the metal as well as the necessary later corrosion introduced sources of difficulties and error, particularly for the comparative study of finer and very tortuous vessels, such as are found in chronic productive inflammations of parenchymatous organs, *e.g.*, contracted kidney. Furthermore, the organs must, often, subsequently be sectioned in order to obtain anything like a comprehensive conception of the arrangement of the arterial tree. The difficulty of doing this without disturbing the metal cast, is apparent when we consider the delicate changes found in pathological conditions.

The use of a wax medium such as employed by Lexer in 1903 and Hildebrand in 1907, instead of metal, is open to the same objections.

The introduction of roentgenography stimulated numerous investigators to apply this method to the study of vascular architecture. Hildebrand credits Dutto with being the first to employ this method in 1896.

The technique consisted in forcing pastes (Opitz, 1897), solutions of the salts of heavy metal suspensions or, in some cases, metallic mercury (Glew, 1899) into the blood-vessels with subsequent roentgenography. This was applied to the study of the coronary arteries by Freyett in 1905 and was considerably perfected by Jamin and Merkel in 1907.

Numerous investigators have employed this method, many of them modifying the medium, using different salts, colloidal silver (Skinner, 1912), pigments (Miller, 1918), etc., and bases

which consisted of oils (Stegmann, 1905-6), paraffin (Hauch, 1903), starch (Hewson, 1915), water (Smith, 1918), gelatines, etc.

The results of these methods were extremely unsatisfactory, since the pictures obtained lacked the plasticity and appreciation of the three dimensions so necessary to the full understanding of circulatory architecture. This fault was to a great extent eliminated by the introduction of the stereoscopic method of roentgenography. Although many important contributions were made in this way, they were all exposed to criticism by admitting too much possibility of artefact in the technique. The results depended greatly on individual skill and experience, and, as no accurate method existed of estimating the mechanical force required in the injection, i.e., pressure exerted on syringe, temperature, viscosity of the medium, etc., comparative studies were unreliable and practically useless.

The old method of injection with pigmented gelatine and of subsequent serial sections with reconstruction was of great value. Its application, however, was limited to the study of the microscopic circulation. Where it was attempted to apply it to the reconstruction of the coarser circulation, individual interpretation was the cause of great diversity of opinion and error. A modification of this method, however, allowed its application to the study of the larger blood-vessels. The injected organs were dehydrated and immersed in an oil which is able to penetrate the tissue thoroughly and give it a uniform refractive index similar to the fluid in which it is placed. In this way the organ was rendered transparent, so that the injected vessels stood out prominently from the cleared parenchyma. The plasticity, so obtained, eliminated many sources of error. The objections to this method, however, were

again those of individual differences, lack of standardization of technique, as well as the inability to render thick organs sufficiently transparent to allow complete visualization of the whole circulatory tree. Nevertheless, to this method we owe the excellent observations made by Spalteholz on the heart (1907). He employed a chrome-yellow gelatine with subsequent clearing.

B. TECHNIQUE EMPLOYED IN THESE STUDIES

As all these methods had their good points and since each had its especial application, the author has used a combination in somewhat modified form.



FIG. 1.—Flanged glass nozzle.

By employing a simple apparatus in which every element of technique could be accurately measured and standardized, uniform injections were obtained with a barium sulphate suspension in gelatine (*vide infra*). After injection, the hearts were roentgenographed stereoscopically, cleared, and finally dissected and sectioned.

A separate series of hearts was injected for the purpose of following out by serial section the minute circulatory structure.

The apparatus used (Fig. 3) has proven eminently satisfactory, so that its uniform results rendered comparative work thoroughly reliable.

The hearts are injected forty-five hours after death to allow rigor to pass off. The chambers are previously washed

free from clot and cannulae inserted into the orifices of the coronary arteries.

In the case of large hearts, large cannulae are used. These are made by coating an ordinary pin with graphite, inserting this into the point of a glass nozzle, heating the tip until red and then flattening down on a hard smooth surface. The pin, which meanwhile has preserved the lumen, can now be withdrawn. In this way a flange is formed at the end of the nozzle (Fig. 1).

In the case of infants' hearts, cannulae are made by soldering a thin rim of silver around a No. 20 gauge 2-inch hypodermic needle and filing off the point beyond it (Fig. 2).



FIG. 2.—Flanged metal needle.

The loose cellular tissue on the external surface of the aorta just at the point of emergence of each coronary is bluntly dissected with a pair of hemostatic forceps until the whole circumference of the vessel is cleared. A silk thread is now tied around the artery as close as possible to the point of emergence, the flange of the needle being below the loop.

Rubber tubing is placed on the cannulae and these are connected by a "Y" tube.

The heart is then suspended by inserting a glass rod under the bridge of the pericardium which lies between the great vessels and the auricles, and resting this rod on a tripod.

The tripod is placed in the upper chamber of the apparatus which consists of a wooden box lined with copper. Separating

lower chamber, which is immersed in a bath kept warm by an electric immersion heater. From here, it is conducted into the upper chamber into a small Wolff flask which contains a thermometer. The temperature of the saline is thus accurately determined before it is forced into the blood-vessels.

By raising or lowering the spiral tube in the water-bath, the temperature can be altered to any degree desired.

The saline at the proper temperature is now forced through the coronary arteries until the washings come clear.

The whole incubator is kept at uniform heat by another immersion electric heater, a thermometer being placed in each chamber. Electric bulbs and glass doors render it quite easy to observe the progress of the injection.

When the blood-vessels are washed quite clean, the pressure tube from the manometer bottle is attached to a Wolff flask containing the injection mass which has been kept in the lower chamber. The mass is forced into the vessels until the mercury in the manometer remains constant at 150 mm. without requiring additional compression. The vessels are then tied off, the cannulae removed, and the heart immersed in cold water.

A suction apparatus attached to the tap is now used to suck out and wash the chambers of the heart free from any accumulated injection mass.

The heart is then immersed in cold formalin until fixed and bleached when it is ready for stereoscopic roentgenograms which should be taken with a tube of medium spark gap. (Dr. Morgan employs the following roentgenographic technique: Tube distance, 24 inches; stereoshift, $2\frac{1}{2}$ inches and 4° ; tube, 4-5 inch spark gap; time, $\frac{1}{10}$ to 1 second exposure according to size; duplitized films, not screened; 20 m. a.

Dr. H. H. Cheney suggests that in very difficult cases where the shadows to be cast are very fine, the best results would be procured by a technique which calls for longer exposures and a very small amount of x-rays.)

For clearing, the heart is gradually dehydrated in alcohol, commencing with 50 per cent and changing every alternate day into an alcohol of 10 per cent additional strength until absolute. After the heart has remained two days in absolute alcohol, the concentration of the latter is tested with an alcoholometer, and if the reading is below 99 per cent another change into absolute is made until finally the reading remains between 99 and 100 per cent. Methylated spirits have proven quite satisfactory for the dehydration.

The heart may now be immersed in synthetic oil of wintergreen. Clearing commences almost immediately. On account of discoloration it may be necessary to change into a fresh quantity of the oil. In two to three days the organ is rendered quite clear and excellent photographs can be taken of the specimen completely submerged in the oil of wintergreen. (For this purpose, use panchromatic plates with an amber filter.)

Dissections are in this way rendered simple, since practically all branches and even branchlets are quite visible and can be followed out minutely.

BARIUM SULPHATE GELATINE. The injection mass employed in this method is made up as follows:

Soak 300 gm. of a fine French gelatine (preferably Gold Label) for two hours in 1,200 c.c. of distilled water. To this add 1,000 gm. of finely powdered barium sulphate, 500 c.c. of distilled water and 2 gm. of thymol. Heat over a water-bath until the gelatine dissolves and stir until the whole mass

becomes a homogeneous milky fluid. Filter through two layers of Victoria lawn.

If pigmented gelatines are to be used, the same technique is employed. The gelatine is made up as follows:

CARMINE GELATINE. Allow 100 gm. of gelatine to soak for two hours in 200 c.c. of distilled water. Warm over a water-bath until the gelatine dissolves and filter through two layers of Victoria lawn. It is advisable to place the filter in an incubator to prevent the jelly from setting while filtering.

Dissolve 15 gm. of aqueous soluble carmine (Grubler's, if possible) in 300 c.c. of distilled water. Slowly add 6 c.c. of ammonia, stirring continually. Filter through two layers of Victoria lawn.

Now mix the filtrate with the filtered jelly. Add about 4 c.c. of acetic acid to the mixture, or to be exact, an amount which by previous titration has been found just sufficient to neutralize 6 c.c. of ammonia. Add 1 gm. of thymol.

Filter once more through two thicknesses of Victoria lawn. With this medium, capillary injections can be easily accomplished and frozen serial sections can be quite successfully obtained. These are mounted in Farrant's solution.

PRUSSIAN-BLUE GELATINE. Proceed as with carmine gelatine, substituting 15 gm. of soluble Prussian-blue for the carmine. It is not necessary to render alkaline and neutralize. (On the whole this has proven less satisfactory than carmine gelatine.)

Bayne-Jones suggests that in order to obtain a thorough penetration of the gelatine into the capillaries (*e.g.*, those in valves), it is necessary to tie off all the cut edges of the heart, as well as the mouth of the coronary sinus. He uses a 1½ per cent carmine solution in gelatine warmed to 45° C. and in-

jects at a pressure of 140 to 190 mm. Hg., the heart being immersed in a salt solution at 50° C.

Where it is desired subsequently to cut sections of rapidly autolysing and changing tissue, *e.g.*, nodal tissue, or where it is required to avoid waiting two days for rigor to pass off, a 10 per cent solution of potassium sulphocyanide (as recommended by McCordick) is allowed to trickle through the coronaries for about one hour. In this way, paralysis and dilatation of the vessels quickly occur and the organ can immediately be injected. Similarly, for roentgenography it is not necessary to fix the tissues, but the organ should be placed into ice-cold formalin until the gelatine is set.

In order to inject the veins of the heart, two methods may be employed: one is to place a cannula into the sinus coronarius; the other, by means of which excellent injection of the *venae parvae* and *venae minimae cordis* may also be obtained, is to tie a cannula into the superior vena cava, tie off all other exits from the right side of the heart, inject directly into the right auricle, freeze the gelatine and carefully remove the contents of the chambers. In venous injection, a relatively low pressure should be employed.

In order to obtain contrasting colors for arteries and veins or between both coronaries, carmine gelatine may be added to the barium suspension in the proportion of 1 to 10.

CHAPTER II

THE BLOOD SUPPLY TO THE VENTRICLES AND AURICLES

IN commencing a description of the distribution of the coronary arteries to the auricles and ventricles, the author is at once confronted with the difficulty of describing a structure which varies not only in different hearts, but also, as we shall see later, in the different age periods.

In order to overcome these difficulties it has been decided to give a description of a theoretical heart whose ventricular and auricular supply represents an average reconstruction from a study of one hundred normal specimens.

Since the variations are in themselves of considerable anatomical interest, however, both physiologically and pathologically, statistical analysis of the hearts will also be given.

The description which follows is divided into two groups: (A) Superficial Divisions; which represent the main branches with their subdivisions usually to the first, second and third order. In this part of their course they are superficial and can easily be seen under the pericardium. After this, however, they penetrate the bulk of the heart musculature in a manner which will be described later, and are known as (B) the Deeper Divisions.

A. SUPERFICIAL DIVISIONS

Figure 4 is a roentgenogram of an approximately average heart.¹ It is seen that it is supplied by two coronary arteries, a right and a left.

¹The roentgenograms reproduced in this monograph give a view of the cardiac circulation as if seen from the back so that one looks through and through the arterial tree. The right side of the plate represents accordingly the right side of the heart and the left side of the plate represents the left side of the heart.

The *right coronary artery* arises from the aorta slightly below the level of the right anterior aortic cusp. From here it proceeds directly to the right and emerges on the anterior surface between the roots of the aorta and pulmonary arteries. Hugging the auriculoventricular sulcus, it rounds the margo acutus, passes that situation of the heart posteriorly where the auricles and ventricles meet, known as the crux, and finally terminates halfway between the latter and the margo obtusus. Here it descends about two-thirds of the way down the posterior surface of the left ventricle in perpendicular fashion.

In its passage around the auriculoventricular groove, the coronary artery is called the *Arteria circumflexa dextra*. It is occasionally bridged by strands of heart muscle. Throughout its course it gives off branches to auricles, ventricles and adjacent fat.

The first ventricular branch is a delicate arcuate twig which is constantly found crossing the root of the pulmonary artery to anastomose with a similar branch from the left coronary artery (Fig. 24). Its site opposite the insertions of the pulmonary cusps defines accurately the junction of the conus with the pulmonary artery and may be of some embryogenetic significance.

On the anterior surface of the right ventricle, two main branches descend to the lower third of the heart. In their course they give off a number of more or less parallel twigs which extend horizontally to the left as far as similar branches from the ramus descendens of the left coronary artery and anastomose with them. Horizontal twigs also extend to the right as far as the margo acutus.

A constant stout lateral branch (*Ramus lateralis*) de-



FIG. 4.—Roentgenogram of the blood supply in the average heart.

scends along the margo acutus towards the apex. At the lower third of the ventricle it often turns posteriorly and extends transversely across the posterior surface of the right ventricle to reach the posterior interventricular sulcus.

The next constant branch to be given off is the *ramus descendens posterior*. This takes origin at the crux and descends along the interventricular furrow posteriorly until it reaches the lower third of the ventricle (Fig. 25).

The terminal portion of the right coronary artery now divides into two or three branches (*Rami ventriculares sinistri*), which descend on the posterior surface of the left ventricle.

The *left coronary artery* arises from the left anterior sinus of Valsalva just a little below the level of the free edge of the corresponding aortic cusp. Proceeding directly forward and to the left, it soon bifurcates under cover of the left auricular appendage. One large and constant division, the *ramus descendens anterior sinister*, is seen emerging on the anterior surface of the heart between the root of the pulmonary artery and the left auricular appendage. Descending along the anterior interventricular furrow, it rounds the apex and ascends the posterior interventricular furrow to reach the lower third of the ventricles. Throughout its course this branch supplies numerous transverse twigs to the anterior surfaces of the right ventricle, as well as large branches which course diagonally down and around the margo obtusus (*Rami marginales*) and maintain a more or less parallel relationship to one another.

Of the latter, one branch is very constant and characteristic in its course, though not so fixed in its origin. Arising generally from the *ramus descendens anterior sinister*, sometimes from the fork between it and the second main division

of the left coronary artery, and less frequently from the latter, it is the most prominent of this group of lateral branches and its terminal divisions sink into the musculature as they reach the posterior surface of the left ventricle.

The second main division of the left coronary artery (*Ramus circumflexus sinister*) lies in the auriculoventricular sulcus and emerges from beneath the left auricular appendage to become superficial where the margo obtusus gives way to the posterior surface of the left ventricle. Here it takes a more or less abrupt downward bend, supplying in its arborizations the upper posterior portion of the left ventricle in a fashion similar to the other rami marginales on the margo obtusus of which it forms another branch.

B. THE DEEPER DIVISIONS

The description thus far given takes no account of many other important branches and divisions. Of these, one of the most important groups is that of the deeper branches.

With the exception of the ramus descendens anterior and posterior, all the other large coronary branches display their main divisions on the anterior surface of the heart. If one were to pierce the heart with a knitting needle from its apex to a point corresponding to the center of the base, generally speaking, the main coronary subdivisions of the second and third order could be said to arrange themselves in a circular manner around the external surface of the heart transverse to the needle as an axis.

From this point on, the finer arborizations are dichotomous and soon plunge into the heart muscle in a direction at right angles to the surfaces of the ventricles. As the ultimate arborizations approach the internal surface of the heart they abruptly

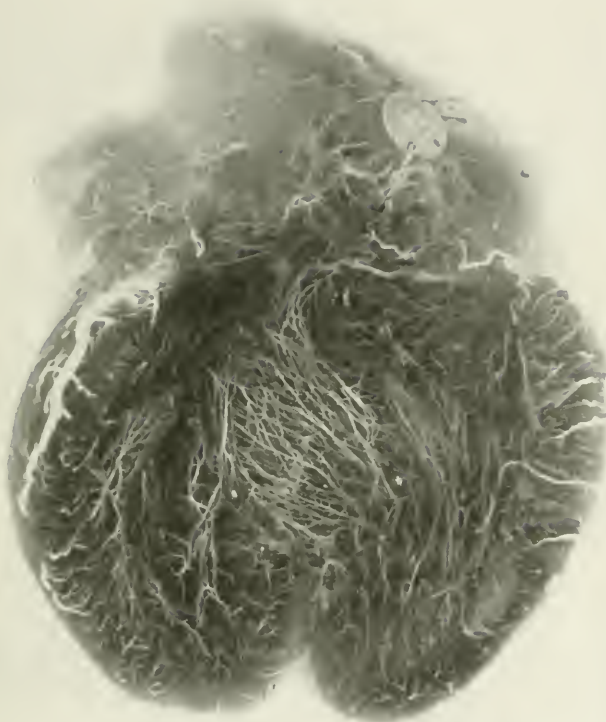


FIG. 5.—Photograph of injected and cleared specimen, showing the ultimate subendocardial distribution of the coronary arteries.

resume a course parallel to it, and, in richly interanastomosing meshes which on the whole have a longitudinal arrangement and tend to follow the intricacies of the columnae carnae, spend themselves macroscopically under the endocardium (Fig. 5). The ultimate capillary distributions follow the direction of the muscle fibers, so that long parallel delicate capillaries lying in the interstices between the individual fibers show here and there a short crossing and connecting link of capillary whose length corresponds to the width of a fiber. We have not found with Meigs that capillaries penetrate through muscle fibers, but that, as held by Köster, capillaries surround individual muscle fibers.

The papillary muscles are turgid with delicate vessels which penetrate them through and through, preserving throughout a longitudinal arrangement with stepladder-like anastomoses. These mount to the apex of the muscle both in its main mass as well as on its surface, as described by Ribbert.

The two rami descendentes of the coronary arteries, as already indicated, have a somewhat different distribution. In addition to the branches which they distribute on the surface of the heart and which divide and subdivide in a fashion similar to other branches, they possess divisions which penetrate directly through the interventricular septum (*Rami interventriculares*), arranging themselves more or less in the manner of harp strings (Fig. 4), those from the ramus descendens posterior dexter spreading out anteriorly, and anastomosing with similar branches from the ramus descendens anterior sinister.

The general arrangement of these branches is parallel with the base of the heart.

The subdivisions from these follow in their ultimate dis-

tribution the same course and character as described for those of the ventricular wall.

It is noteworthy that the topmost of these rami inter-ventriculares (*Ramus septi fibrosi*) arises from the ramus circumflexus dexter at the crux, proceeds anteriorly along the region between the interventricular and interauricular septa and supplies, in its finest ramifications, the tissue in and around the opening of the coronary sinus (Fig. 8). It extends from this situation anteriorly, anastomoses with a corresponding branch from the left coronary artery and penetrates through the central fibrous body and undefended space (*Pars membranacea septi*).

The *auricular branches* of both coronary arteries do not show as constant an arrangement as the ventricular supply, nevertheless, there are certain characteristic features worthy of note.

There are, as a general rule, two auricular branches which arise from the anterior portion of the arteria circumflexa dextra (*Rami anteriores*). The first of these arises as a stout branch very soon after the origin of the right coronary artery and, coursing at once posteriorly, reaches the anterior wall of the right auricle and follows this as it curves behind the aorta, maintaining a horizontal position about half a centimeter above the auriculo-aortic groove (Figs. 4 and 8). Reaching the site of the interauricular septum, it gives off several twigs which surround the upper portion of the right auricle anteriorly and supply the appendage and adjoining regions, whereas the main vessel turns abruptly posteriorly and passes through the septum to reach the posterior portion. Here, it turns, and, ascending the superior wall of the right auricle posteriorly to the cava insertion, it surrounds in ring fashion

the site of junction of the superior vena cava with the auricle (*Sulcus terminalis*) and arborizes extensively in the tissue in and around the auriculoventricular node.

Throughout its course, this vessel gives and receives numerous anastomosing twigs with the other auricular branches. As will be shown later, it may arise from the left coronary artery, in which case its course is as follows:

Arising very close to the origin of the left coronary artery, often from its posterior main division, it either encircles the base of the left auricular appendage, ascending the external surface of the left auricle and crossing directly to the region of the superior vena cava where it terminates in a manner similar to that described for the right coronary origin of this vessel, or it passes back immediately after its origin so as to course along the anterior wall of the left auricle about half a centimeter above the auriculo-aortic groove. When it reaches the site of the interauricular septum, it turns up and crosses on the superior aspect of the right auricle, between the superior cava funnel and the right appendage, ultimately to attenuate itself around the opening of the inferior vena cava in a manner which is described in greater detail in Chapter IV.

Throughout its course, it too, gives off numerous anastomotic twigs, and, particularly as it crosses the roof of the right auricle, gives numerous arborizations to the seat of the sino-auricular nodal tissue.

This most constant and probably most important auricular branch shows so characteristic a course that it merits the name of *Ramus ostii cavae superioris*.

The second anterior branch to the auricles from the right coronary artery is less constant. It ascends the aortic face of the right auricular appendage and distributes itself over the

roof of the right auricle, approaching the origin of the superior vena cava.

Very occasionally a lateral branch is given off at the margo acutus, which ramifies over the external surface of the right auricle, approaching the opening of the inferior as well as that of the superior vena cava.

A number of very small and inconstant auricular vessels may be seen besides those described above, but since there is nothing characteristic about these and since they are with difficulty distinguishable from vessels which supply the pericardial fat, the description of these will be taken up under the heading of *Arteriae telae adiposae cordis*.

The left coronary artery shows, with the exception of the ramus ostii cavae superioris already described, one or two smaller auricular branches which distribute themselves over the anterior surface and roof of the left auricle. Occasionally a left lateral auricular branch extends around the posterior surface of the auricle to reach the opening of the superior vena cava.

ARTERIAE TELAE ADIPOSAE CORDIS

Besides the main vessels which have thus far been described, there are a number of interesting and important branches which apparently occupy both anatomically and functionally a category of their own, because considered from both these viewpoints they seem to occupy a place half way between that of the vasa vasorum and the cardiac coronary branches proper.

These are what may be called fat branches (*Arteriae telae adiposae*). They are seen in greatest number in the fat found under the pericardium—namely, in the grooves between

the chambers, where they occur as an irregular felt-work, and over the sites of the main coronary branches, where they exist as delicate parallel accompanying vessels whose distance from the main branches varies directly with the amount of fat present (Figs. 24 and 25). The rich interanastomosing network of delicate vessels, superimposed upon the outer coats of the root of the aorta and of the pulmonary artery, falls under this category. These fat-vessels arise largely from the first portions of the *arteria circumflexa sinistra* and *dextra* as well as from their branches soon after their origin.

In a later chapter their importance and significance will be shown.

BLOOD SUPPLY TO THE HEART AS A WHOLE

It is of considerable interest and importance to coordinate the description given above of the distribution of the individual coronary arteries and consider the supply of the heart as a whole as well as of its specific parts.

Numerous investigators have attempted to inject both coronary arteries at the same time, and, by using a differently colored injection mass for each coronary artery, have obtained results which in some measure help to throw light on those regions of the heart which are supplied by both.

Amenomiya, Nussbaum and Sternberg have contributed much valuable information in this direction.

It must first of all be remembered that there can be no sharp line of demarcation between the supply of right and left coronary arteries, since, not only do their branches overlap, but also, as will later be shown, profuse and abundant anastomoses leave a wide borderline which is supplied by both vessels. A rather arbitrary division will therefore be given,

first, of the supply of each artery; secondly, of the common supply by both vessels.

Since the auricular distribution of blood-vessels is so prone to variations that an attempt at giving a typical description becomes artificial and practically worthless, this will be left to the consideration of variations. The following description, therefore, answers in a rather unsatisfactory way for the supply to the ventricles.

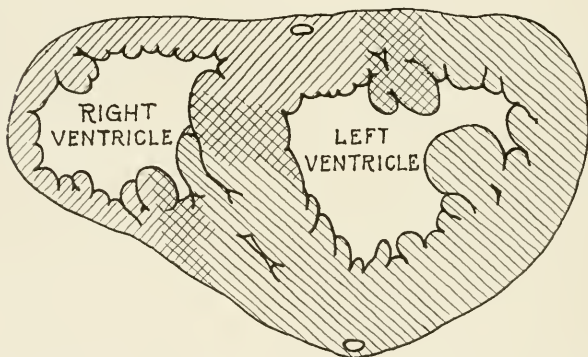


FIG. 6.—Diagram of cross section through the heart, showing regions supplied by the left and right coronary arteries.

Lines running from right to left represent the supply of the left coronary artery.

Lines running from left to right represent the supply of the right coronary artery.

The crossed areas represent supply from both vessels.

From the description which has been given above, it can readily be appreciated that the right coronary artery in the typical average heart supplies the entire right ventricle with the exception of the left third of the anterior wall. Besides this, its rami ventriculares sinistri supply the right half of the posterior wall of the left ventricle and a small strip of the interventricular septum (Fig. 6).

The left coronary artery, on the other hand, supplies the whole remaining part of the left ventricle, the small left anterior portion of the right ventricle not supplied by the right coronary artery and a small anterior strip of the interventricular septum.

The areas of junction on the posterior surface of the left ventricle and on the anterior surface of the right ventricle where these divisions meet, are supplied by both vessels. Thus, the intervening portion of the interventricular septum is supplied by branches from the ramus descendens posterior dexter and ramus descendens anterior sinister and the posterior papillary muscle of the left ventricle by the rami ventriculares sinistri from the right and the rami marginales from the left coronary artery. At least a portion of the anterior papillary muscle of the right ventricle is supplied from the rami interventriculares from the left and the rami anteriores from the right coronary artery. Indeed, it is quite easy to trace a distinct large vessel through the trabecula septomarginalis to this papillary muscle, which appears to supply the greater volume of its blood to this region.

The large anterior papillary muscle of the left ventricle receives its blood from the rami marginales and the inconstant posterior papillary muscle of the right ventricle from the rami posteriores of the right coronary artery.

CHAPTER III

VARIATIONS IN THE DISTRIBUTION OF THE CORONARY ARTERIES

AS has been indicated, the coronary arteries are rather prone to variations. It is this disposition to variations which renders their description somewhat artificial and rigid. There appears, however, to be one *typical* variation which is rather constant and which will be described here. For the reason that the other possible variations assume clinical and pathological interest, a table classifying a statistical analytical study of the course and character of the coronary arteries will follow.

If one makes a review comparing the general characteristics of the average distribution of left and right coronary arteries, one is struck by the fact that the main stout ramus descendens anterior of the left coronary artery has its counterpart in the ramus descendens posterior of the right; furthermore, that the left coronary artery seems to trespass on the anterior surface of the right ventricle and falls somewhat short of supplying completely the left ventricle posteriorly.

The main scheme, therefore, in so far as the ventricular supply is concerned, seems to be a sort of twisting of the vessels in a direction from left to right anteriorly and right to left posteriorly.

An examination of what is termed the *typical* variation (Fig. 7) shows, first of all, that both the anterior and posterior rami descendentes come from the left coronary; secondly, that the rather aborted ramus circumflexus sinister, which is



FIG. 7.—Roentgenogram of a typical variation in coronary artery distribution.

seen in the normal heart, is represented here by a very typical and characteristic circumflex branch which imitates accurately that found normally on the right side. Thus, even the branches, which have been described as originating for the most part from the fork of the two main coronaries normally, in this instance arise from the left coronary artery all along its course through the auriculoventricular sulcus and up to a point where it, again imitating the right coronary artery, abruptly bends downward at the site of the crux and proceeds to the lower third of the heart. The third striking feature is the very much aborted and simplified right coronary artery. The ramus circumflexus, now resembling that normally found on the left side, ends at the margo acutus and gives off some delicate branches, one of which continues in the auriculoventricular sulcus towards the crux, the others presenting a rather scant supply over the posterior face of the right ventricle. The main body of the right coronary turns abruptly down at the margo acutus and finally attenuates itself toward the apex. For the rest, the subdivisions of this vessel on the anterior surface of the right ventricle is similar to that found in the normal.

The auricular supply in this type shows no characteristic variation.

STATISTICAL ANALYSIS OF THE VARIATIONS

The site of origin of the coronary arteries shows considerable variation. Since, as Tandler points out, the old discussion between Morgagni and Fantoni and later between Hyrtl and Brücke, as to the importance of the site of opening of the coronary arteries in connection with the position of the semilunar cusps in systole, has lost its significance, it is sufficient to state with Piquand that they arise in the center

line of the sinus of Valsalva somewhat anteriorly to the insertion of the cusps.

So far as the number of the coronary arteries at their origin is concerned, it is interesting to note that Morgagni, Cruveilhier, Hyrtl, Bochdalek and Engelmann have described cases where the total blood supply to the heart came from a singly existing coronary artery.

Fallopian and Riolanus held that normally one coronary artery exists in the heart. Morgagni, however, showed that two exist.

Occasionally more than two coronary arteries spring from the aorta. In such instances the accessory branch or branches represent twigs which, in the ordinary course of events, spring from the main coronary, but which, in these cases, arise directly from the aorta close to the origin of the main stem.

Tandler states that this multiple origin of coronaries is found on both sides, more frequently on the left. Out of the 100 cases studied by the author, in four instances a second origin of a coronary artery was seen; of these, two were on the left side and two on the right. Here the accessory coronary pursues a course identical with one of the *rami ventriculares anteriores*. The accessory openings in the left anterior sinus of Valsalva were somewhat to the left of the opening of the main coronary artery. Of those on the right side, one arose to the left, and the other, below and to the right of the main coronary artery.

That this condition occurs more frequently than reported and is often missed, seems quite possible, since the accessory coronary branch is usually very small and, unless a careful dissection is made or an injection directly down the lumen of the aorta carried out, the condition can easily be overlooked.

Although occurring relatively infrequently, the condition assumes some clinical importance, for an obliteration of a main coronary artery which leaves intact an accessory twig may enable a heart, which has had time to adapt itself to the diminution in its blood supply, to carry on a relatively undisturbed function.

VARIATIONS IN THE MAIN BRANCHES

In considering the variations occurring in the main branches of the coronary arteries, it has been decided to present these in table form. This can conveniently be expressed by categorizing, according to their site of origin and main course, the branches in the 100 hearts which were very carefully studied, indicating the percentage in which they occurred singly, as two branches, three, etc.

In the text which follows each chart, a more detailed discussion on the qualitative nature of the variations is given.

TABLE I.—ARTERIA CORONARIA DEXTRA

| SUPPLY TO VENTRICLES | | | | | | |
|---|----|----|----|----|----|-------------------------|
| Number of Branches..... | 0 | 1 | 2 | 3 | 4 | 5 |
| Rami Anteriores..... | 4 | 36 | 47 | 13 | 0 | 0 |
| Rami Laterales..... | 6 | 84 | 8 | 2 | 0 | 0 |
| Rami Ventriculares Dextri Posteriores.. | 48 | 40 | 8 | 0 | 4 | 0 |
| Ramus Descendens Posterior..... | 8 | 92 | 0 | 0 | 0 | 0 |
| Rami Ventriculares Sinistri..... | 16 | 32 | 22 | 18 | 10 | 2 |
| | | | | | | Percentage of Incidence |

TABLE II.—TERMINATION OF RAMUS CIRCUMFLEXUS DEXTER

| Margo Acutus | Crux | Between Crux and Margo Obtusus | Margo Obtusus |
|--------------|-------------|--------------------------------|---------------|
| 4 per cent | 10 per cent | 66 per cent | 20 per cent |

Where several rami anteriores exist, they course somewhat diagonally across the anterior surface of the right ventricle and approach the ramus descendens sinister at right angles. In their course they are parallel and relatively equidistant to one another. They do not dichotomise with frequency in their main stems but rather towards the end of their course on the surface of the heart.

Where the rami anteriores occur in smaller numbers, they proceed in a line drawn to the apex, giving off all the while parallel and more or less equidistant branches to the right and principally to the left side in a manner very similar to those described above.

The ramus lateralis, as will be seen in Table I, occurs most frequently singly. Here it descends in somewhat tortuous fashion towards the apex, divides before reaching it and distributes branches to the anterior and posterior aspect of the lower portion of the right ventricle.

The rami posteriores are infrequent. Occurring usually singly, they have a short course parallel to the ramus lateralis.

The ramus descendens posterior terminates in 5 per cent of the cases at the upper third of the posterior interventricular sulcus, in 27 per cent at the middle, in 39 per cent in the lower third, and in 29 per cent at the apex.

When the rami ventriculares sinistri occur multiple, they are given off, as a general rule, fairly close one to the other near the crux, spending themselves in their coarser distribution on the inner and upper half of the posterior surface of the left ventricle.

Of the termination of the ramus circumflexus dexter (Table II), it need only be said that 66 per cent end somewhere between

the crux and the margo obtusus. Of these, 41 per cent end half-way between the two points.

TABLE III.—ARTERIA CORONARIA DEXTRA

SUPPLY TO AURICLES

| Number of Branches..... | 0 | 1 | 2 | 3 | Percentage of Incidence |
|--|----|----|----|----|-------------------------|
| Rami Anteriores..... | 11 | 52 | 26 | 11 | |
| Rami Laterales..... | 62 | 36 | 2 | 0 | |
| Rami Auriculares Dextri Posteriores..... | 79 | 16 | 5 | 0 | |
| Rami Auriculares Sinistri Posteriores..... | 84 | 12 | 4 | 0 | |

As has been pointed out before, with the exception of the stout branch to the junction of the superior vena cava with the right auricle (*Ramus ostii cavæ superioris*), the auricular branches are rather inconstant in their distribution. In a great number of cases the branches are so small that the borderline between those which may be called auricular branches, and those which fall into the category of fat branches, is very uncertain. It is noteworthy that, although both auricular as well as fat branches usually arise from the main ramus circumflexus, they also take origin from the beginning of the ventricular branches.

As will be seen by the nomenclature (Table III), the division of these branches into groups according to their site of origin is purely arbitrary, since this may not, and often does not, correspond with their ultimate distribution. Thus, a ramus auricularis sinister posterior is occasionally seen to cross the region of the interauricular septum from left to right and ultimately to arborize on the right side of the right auricle. Indeed, it is not infrequently found that the vessels pursue a bizarre course and supply at random almost any conceivable portion of the auricles irrespective of the original site of origin.

It is true, however, that to a certain extent a branch will supply that portion of the auricle which it first crosses and in this way there is some correlation between the nomenclature and the function of the vessel.

It is on account of this rather inconstant supply of these vessels that it has been decided to give, at the end of this chapter, an analysis of the blood supply to the auricles in bulk, that is, stating approximately the ratio of auricles supplied from the left and right coronary arteries.

TABLE IV.—ARTERIA CORONARIA SINISTRA

| SUPPLY TO VENTRICLES | | | | | | | |
|---------------------------------|----|-----|----|----|----|---|-------------------------|
| Number of Branches..... | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Ramus Descendens Anterior..... | 0 | 100 | 0 | 0 | 0 | 0 | 0 |
| Rami Marginis Obtusi..... | 0 | 6 | 23 | 45 | 14 | 6 | 6 |
| Rami Posteriores..... | 84 | 8 | 6 | 2 | 0 | 0 | 0 |
| Ramus Descendens Posterior..... | 92 | 8 | 0 | 0 | 0 | 0 | 0 |
| Ramus Ventricularis Dexter..... | 92 | 7 | 0 | 1 | 0 | 0 | 0 |
| | | | | | | | Percentage of Incidence |

TABLE V.—TERMINATION OF RAMUS DESCENDENS ANTERIOR

| Lower Third of Anterior Interventricular Sulcus | Apex | Lower Third of Posterior Interventricular Sulcus | Middle of Posterior Interventricular Sulcus |
|---|-------------|--|---|
| 2 per cent | 38 per cent | 52 per cent | 8 per cent |

TABLE VI.—TERMINATION OF RAMUS CIRCUMFLEXUS SINISTER

| Margo Obtusus | Between Margo Obtusus and Crux | Crux | Between Crux and Margo Acutus |
|---------------|--------------------------------|-------------|-------------------------------|
| 86 per cent | 2 per cent | 10 per cent | 2 per cent |

Here it must be noted, first, that the ramus descendens anterior gives a number of branches to the right ventricle, which run in the same direction and meet the rami anteriores from the right coronary artery. Secondly, the rami marginis obtusi, which are represented on the right side by the rami anteriores and laterales, arise, as pointed out before, somewhere on the left ventricular aspect of the ramus descendens and ramus circumflexus sinister. The variations, here, are characterized by a shifting of their site of origin anywhere from the extent of the ramus descendens to the ramus circumflexus as far as the junction of the margo obtusus with the posterior surface of the left ventricle.

The greater number of these diagonally running vessels come off the ramus descendens, the lowermost terminating their superficial course on the anterior surface of the left ventricle in the region of the apex. The remainder, together with those taking origin from the ramus circumflexus, round the margo obtusus to reach the posteroexternal portion of the left ventricle.

There are usually one or two branches from the ramus circumflexus opposite the center of the margo obtusus, which are larger than the remaining rami marginales and which extend downward towards the apex.

The first characteristic variation to be considered is the termination of the ramus descendens anterior. It will be seen in Table V that the site of election is the lower third of the posterior interventricular furrow. In this regard it is to be remembered that the final, minute termination extends much farther. The place, however, where the superficial and coarser circulation ends has been used as the point of termination.

In 84 per cent of the cases there were no distinct rami

posteriores and in these cases the posterolateral surface of the left ventricle was supplied by continuations of the rami marginis obtusi.

Rami descendentes posteriores occurred only in instances where the ramus circumflexus reached the crux. In the same instances rami ventriculares dextri were given off, usually only one; in one case, three (Table IV).

TABLE VII.—ARTERIA CORONARIA SINISTRA

| SUPPLY TO AURICLES | | | | | Percentage of Incidence |
|--|----|----|----|----|-------------------------|
| Number of Branches..... | 0 | 1 | 2 | 3 | |
| Rami Anteriores..... | 5 | 45 | 27 | 23 | |
| Rami Laterales..... | 77 | 23 | 0 | 0 | |
| Rami Auriculares Sinistri Posteriores..... | 86 | 14 | 0 | 0 | |
| Rami Auriculares Dextri Posteriores..... | 0 | 0 | 0 | 0 | |

The same remarks as to the inconstancy in course and character of the auricular branches, which were made for the rami auriculares from the arteria coronaria dextra, apply equally well to those from the arteria coronaria sinistra.

It will be noted in Table VII that there are no branches arising from that part of the ramus circumflexus sinister which occasionally (in 2 per cent of cases, Table VI) extends beyond the crux to the right side. This, of course, does not mean that auricular branches from the left coronary artery do not extend over to the right side, for this is, as a matter of fact, not infrequently seen.

Upon examining the roentgenograms carefully, it was found that in 44 per cent of the cases most of the circulation to both auricles came from the right coronary artery; in 36 per cent of the cases, the distribution of auricular branches and vascular structure was about equal from the right and left

coronaries, and in 20 per cent of the cases most of the blood came from the left coronary artery.

It is interesting to note, in this connection, what an important rôle the ramus ostii cavæ superioris plays, for in 82 per cent of those cases which showed a preponderance of supply from the right side, there existed a ramus ostii cavæ superioris taking origin from the right coronary artery. In 100 per cent of the cases where the preponderant supply was from the left coronary artery, the ramus ostii cavæ superioris arose from the same side. Taken altogether, the ramus ostii arose from the right coronary artery in 60 per cent of the cases, and from the left in 40 per cent.

From the foregoing description of the variations which occur in the branching and ultimate distribution of the coronary arteries, it follows that the heart as a whole, as well as its parts, *e.g.*, septum and papillary muscles, presents functional differences corresponding to these variations, an observation which is rather important clinically and anatomically, for, whereas for example, in the *typical* blood supply an infarction and necrosis of the posterior papillary muscle of the left side would indicate a closure of branches from both sides, in the *typical variation* which has been described, closure of one or two rami posteriores from the left coronary alone would cause the same condition. Again, when the ramus circumflexus dexter reaches the margo obtusus (20 per cent), obliteration of it alone would lead to a total necrosis of that muscle; as also with the supply to the interventricular septum.

The most frequent seat of variations, so far as supply from left or right is concerned, lies in the extent of supply to the posterior wall of the heart. As has been noted, this may vary

from cases where branches from the left coronary artery extend practically to the margo acutus, to where branches from the right coronary artery extend to the margo obtusus. In these instances the region of the heart supplied by each, as well as the region of the heart receiving blood from both, will vary with the anatomy of the circulatory structure.

CHAPTER IV

THE BLOOD SUPPLY TO THE NEUROMUSCULAR TISSUE

IN describing the blood supply to the conducting apparatus of the heart, to which, among others, notable contributions have been made by Purkinje, Gaskell, Kent, W. His, Jr., Keith and Flack, Aschoff and Tawara, Moenckeberg, Koch, Fahr, Hering and Erlanger, it is deemed advisable to give a short description of this structure in order to review its morphology and, therefore, appreciate better the specificity of its vascular architecture as well as the functional significance of the latter.

This structure which, despite the objections by some observers, as Dogiel, is now held to be responsible for the orderly origin and conduction of impulses from auricles to ventricles, consists of: (a) two main nodes which are, according to Thorel, united by a specially differentiated strand of tissue, but, according to Keith and Flack and Koch, ununited except by the ordinary musculature of the auricles; (b) a main auriculoventricular conducting bundle which divides into two limbs for right and left ventricles; (c) a very rich and profuse arborization of the limbs in both ventricles.

Of the two main nodes, one, known as the *sino-auricular*, lies in the sulcus at the junction of the superior vena cava with the right auricle, more on its anterior aspect. The other, known as the *auriculoventricular* node, lies within the mouth, and extends to the left of the opening of the sinus coronarius into the right auricle. It is certain that the main conducting or

His bundle is a direct continuation of the latter, which, lying in a sheath (described by Curran) penetrates the septum fibrosum. This is from 1 to 3 mm. in width and 1 to 2 cm. in length and usually runs along the lower border of the pars membranacea septi where it divides into a right and left septal branch which saddle the top of the interventricular septum. The right limb extends into the vestigial moderator band (*Trabecula septomarginalis*, Tandler) as a fairly distinct column which may or may not course superficially. Here it breaks up into an extensive interlacing and interanastomosing network of fibers which are generously distributed over the internal surface of the right ventricular wall.

The left limb passes over the top of the interventricular septum and, soon spreading into a fan-shaped, thin and flat structure, distributes itself over the interior surface of the left ventricle in two divisions. These are called, according to their location on the interventricular septum, fasciculus anterior and fasciculus posterior.

So far as the histology of this structure is concerned, it may be stated briefly that the nodes and main bundle are made up of a meshwork of narrow, somewhat fusiform fibers with more or less indistinct striations. These cells gradually give way, as they ramify in the ventricles, to large pale fibers of relatively undifferentiated protoplasm with striations clearly seen in their outer strata. The nuclei of the main bundle are numerous and always surrounded by a perinuclear space; those of the ultimate ramifications never occur together and are always near the border of the cells.

In 1907 Keith and Flack first pointed out that the sino-auricular node possesses a distinct and specific blood vascular system. This was substantiated in 1909 by Koch, who described

in greater detail a stout branch from the right coronary artery, which courses between the aorta and mesial wall of the right auricle to penetrate the interauricular septum and sends a twig to surround posteriorly the cava sulcus. Here it anastomoses with a delicate auricular branch which arises from the main coronary in this situation and ascends between the musculae pectinati, passing practically free through the interstices. The vessels now pass the situation of Wenckebach's bundle and pierce through the sino-auricular node.

In 1906 Tawara described a special blood supply to the auriculoventricular node, the main bundle and its chief divisions. This was later described by Keith and Flack as a relatively large branch of the right coronary artery which accompanies the main bundle as it penetrates the annulus fibrosus.

Moenckeberg (1908) described vessels supplying the auriculoventricular node and the main bundle but did not consider them specific or constant in their supply. He had seen a thick vessel emerging from the auricular septum to enter the bundle and divide into many small vessels which accompany the stem of the bundle forward. From these, two branches continue to the anterior portion of the bundle up to the point of division.

Spalteholz (1909) observed a delicate vessel entering the bundle but was unable to determine its ultimate course.

In 1910 Georg Haas published the results of his researches in this direction. He employed differently colored injection masses for each coronary artery, filling, in some cases, a single coronary artery; in others, both at the same time.

Isolating what he believed to be the specific branch to the auriculoventricular nodal system, he also attempted to inject this branch alone, but was unsuccessful on account of technical difficulties.

Haas's experiments consisted in injections, dissections and serial sections of human, dogs' and calves' hearts, and from these he came in part to the following conclusions:

In man the right coronary artery plays the chief rôle, supplying two branches from the posterior coronary arch. First, a *ramus septi ventriculorum superior*, which supplies the upper posterior half of the septum, piercing into the left ventricle and supplying the posterior divisions of the neuromuscular limb in this situation. Secondly, a *ramus septi fibrosi* which, coursing through the auricular septum, sends several branches through the septum fibrosum to the inner muscular layers of both ventricles and finally, as a stout twig, enters Tawara's node and loses itself in the main bundle and beginnings of both neuromuscular limbs. The anterior branches to the left main limb are supplied by fine twigs from the left coronary artery; the right limb lies just on the border between the regions of arborization of the right and left coronary arteries in the septum.

He is of the firm opinion that in the human heart, no anastomoses exist between the right and left coronary arteries in the auriculoventricular node and main bundle.

From a study made on 100 hearts, the author is of the opinion that a distinct and specific blood supply exists for both sino-auricular and auriculoventricular nodes, the main bundle, the first portion of the left limb and a large part of the right limb of the neuromuscular system. The remainder shows a supply which corresponds to the area of heart musculature upon which it rests.

The discrepancies in the otherwise excellent work of Haas are probably due to technical errors, Haas himself admitting difficulties. Dissections in an uncleared specimen, even though perfectly injected, are at best open to error, and serial sections, though throwing much light on the microscopic appearance of capillaries, etc., are eminently unreliable for coarser reconstruction.



FIG. 8.—Photograph of injected and cleared specimen, showing the blood supply to the neuromuscular tissue on the right side.

Figure 8 shows an injection by the method described by the author in the chapter on technique. Here it is seen quite clearly that a very stout and characteristic branch which arises close to the origin of the right coronary artery, in this case makes a fairly direct course for the region of the sinoauricular node and is called, on account of its obviously specific supply, the *Ramus ostii cava superioris*. As its detailed description has already been given (Chapter II), it need only be pointed out that though this vessel is more or less characteristic in its ultimate supply, it is not absolutely fixed in its origin, for it may arise from either left or right coronary artery. Furthermore, the author has been unable to find the characteristic twig which Koch describes as arising from the main coronary close to the origin of the inferior vena cava and passing practically free through the interstices of the musculæ pectinati, but has found instead:

First, that *several* auricular branches in the vicinity give off numerous anastomotic twigs; secondly, the ramus ostii cavae superioris shows extensive anastomoses with its own branches; thirdly, in certain instances where the ramus ostii cavae superioris arises from the left coronary artery (not pointed out by Koch) it may either supply the nodal tissue only as it passes this site anteriorly, other auricular branches contributing largely to the ring-shaped encircling arterial structure, or, it may bifurcate as it reaches the cava funnel and encircle it with two embracing branches which anastomose freely with each other as well as with other auricular branches in the region of the external surface of the right auricle; fourthly, the author has failed to find a vessel which lies, on the whole, free in the interstices of the musculæ pectinati.

There are never two rami ostii cavae superiores, always

one. No blood-vessels have been found to show a supply for a specific tissue which might connect the sino-auricular with the auriculoventricular node, an observation which is confirmatory of Koch's.

Figure 8 also shows the *ramus septi fibrosi*. This corresponds, except for a few details, very well with the description given by Haas. It arises invariably from the crux, and since this is crossed most frequently by the ramus circumflexus of the right coronary artery, it receives its blood usually, though not invariably, from this source.

Coursing directly anteriorly it gives branches to the neighboring tissues, one of which, usually a stout twig, is fairly constantly seen supplying the superior portion of the interventricular septum. The main (though occasionally more delicate) vessel, however, plunges into the auriculoventricular node and accompanies this as it gives way to the main bundle, and often the bundle in its primary divisions.

Just as the *ramus septi fibrosi* represents really a superior septal branch from the *ramus descendens posterior*, even so its behavior is similar to these, inasmuch as it receives anastomoses from the superior septal branches of the left coronary artery.

These septal branches can readily be seen in a cleared specimen. They curve close under the insertion of the right aortic cusp, in the musculature of the interventricular septum, sometimes better seen on the left side, and approaching the undefended space, ramify in the neuromuscular tissue in the septum fibrosum, anastomosing with the corresponding vessel from the right side.

The main point of difference in the circulation as described here, with that of Haas, lies in the existence of anas-

tomoses which take place between branches from the left as well as the right coronary arteries.

In a case cited by Haas this anatomical construction is borne out. This concerns a heart where there occurred embolization of the ramus septi fibrosi with hemorrhagic infarction of the interauricular septum and the muscular divisions which enter into the nodal structure. The anterior portion of the node was preserved though much altered by inflammation. Here, Haas states, perhaps anastomoses occurred with the ramus superior septi ventriculorum or with the anterior septal artery.

The *right* limb of the neuromuscular bundle, as has been stated, pursues a practically uninterrupted course through the trabecula septomarginalis. Throughout, it is accompanied by a stout vessel (*Ramus limbi dextri*) derived from one of the earliest divisions of the ramus descendens sinister, and really representing a septal branch. At the base of the trabecula, twigs from the rami anteriores of the right coronary artery penetrate and anastomose with the ramus limbi dextri.

The ultimate arborizations are supplied from the rich subendocardial vessels which have been described on page 16, and correspond in their nutrition with the area of heart musculature upon which they lie.

The *left* limb of the neuromuscular structure has no specific blood supply of its own, but derives its nourishment on its septal aspect from the profuse anastomoses of septal branches from both sides, the fasciculus anterior receiving more blood from the left coronary artery, and the fasciculus posterior, from the right. For the rest, here, as in the right ventricle, the ultimate divisions vary in their blood supply according to their situation on the heart musculature.

It is important to remember that variations in the vascular architecture will profoundly alter the blood supply to the neuromuscular tissue.

It has already been pointed out that the sino-auricular node is supplied from the right coronary artery in 60 per cent of the cases examined, and from the left, in 40 per cent. Since the ramus septi fibrosi arises from that coronary artery which passes the crux, it follows that its origin is as follows:

- (a) Right Coronary Artery.....86 per cent
- (b) Left Coronary Artery..... 4 per cent
- (c) Possible from left or right.....10 per cent

The percentage of possibilities from left or right is due to the fact that this figure represents the proportion of cases where both left and right ramus circumflexus reach the crux. In these cases, 6 per cent of the possibilities came from the right and 4 per cent from the left, so that the final figures are:

- (a) Right Coronary Artery.....92 per cent
- (b) Left Coronary Artery..... 8 per cent

As with the ramus ostii cavae superioris, this branch always arises from one side, never from both.

Since no variations were found in the occurrence of the rami interventriculorum from the ramus descendens anterior sinister, at least in our cases, it may be assumed that the limbus dexter of the neuromuscular tissue invariably derives its blood from the left coronary artery.

So far as the blood supply of the fasciculi of the limbus sinister on their septal aspect is concerned, in those cases where the ramus descendens posterior arose from the left coronary artery (8 per cent) they were supplied wholly from

this artery. This, as will be noted, corresponds to the ratio of supply to the auriculoventricular node from the same side. In the other 92 per cent, both coronary arteries contributed to their supply.

The ultimate distribution of the Purkinje fibers also corresponds, as regards their blood supply, to the variations described in Chapter III.

To bring out the clinical and pathological importance of this vascular arrangement, as well as to lend support to the anatomical description, there follows a series of cases described by Moenckeberg, in which distinct pathological lesions of the neuromuscular structure are traced to interference with its specific blood supply.

It is already well known that this tissue at times shares pathological processes with the rest of the heart musculature; at times escapes; and, at times, is the sole part affected in generalized hematogenous disturbances.

Moenckeberg, for example, has shown that in a case of endo- and focal myocarditis, a distinct infiltration and congestion of the neuromuscular tissue occurred.

In some cases of brown atrophy, the bundle contained fat and much more brown pigment than the muscular tissue proper.

One case of arteriosclerosis and brown atrophy of the heart showed an absence of pigment in the limbus sinister.

In a male, aged seventy-seven, who died of pyelonephritis, heart failure and brown atrophy of the heart, the bundle was very fatty but contained no brown pigment.

Moenckeberg attributes this discrepancy in the pigmentation of the bundle to the fact that pigmentation must be advanced before it affects the bundle.

In another case dying from general peritonitis, fatty rings were found only in the bundle. It is to be remembered that normally the bundle contains fat.

In a case of thoracic mesaortitis with stenosis of a coronary artery, there was no necrotic lesion in the bundle. This case assumes particular significance when, by contrast, a series is examined where this bundle artery has been blocked. Thus, in a male, aged eighty-two, who showed clinically the Stokes-Adams syndrome with bradycardia and dissociation, and who presented at autopsy a heart with concentric hypertrophy of the left ventricle and considerable sclerosis of the main coronary arteries with, however, patent lumina, a scar was found at the site of the bundle. As Moenckeberg could find no other explanation for this, other than embolic blocking, he came to the conclusion that "this condition is, however, only to be explained by assuming a distinct and specific artery to the bundle, and in this way the above case supports the view that the blocked bundle must have contained a specific vessel."

In Case xxiv of Moenckeberg's series, there is an interesting illustration of the specificity of supply to the bundle. In an individual, aged sixty-three, who died with arteriosclerotic kidney, there was found arteriosclerosis of the coronaries with old closure of the right and many scars in the heart muscle. *The septum in the region of the bundle was scarred, but the bundle itself was intact.* Here evidently the vasculature to the bundle was functioning perfectly.

That scarring may be scattered in the bundle so that some of the fibers are spared, is illustrated in Case xxv. This concerns a male, aged seventy-five, who died of heart block, general arteriosclerosis and atheroma of the coronary arteries. Here, there was found scarring at the origin of the bundle but

a number of fibers passed through intact. Evidently the blocking was only in some of the finer divisions of the ramus septi fibrosi.

Additional *a posteriori* evidence of the existence of a specific bundle blood supply is found in Case xxvi. A male, aged fifty-seven, who showed at post-mortem coronary arteriosclerosis, chronic heart aneurysm, old mitral endocarditis, etc., presented an intact bundle lying on an extensively and thoroughly scarred subjacent and surrounding tissue. Case LIX showed, similarly, anemic necrosis of the septum with the bundle intact.

So far as the effect of coronary artery obliteration on the *ultimate* distribution and function of the neuromuscular structure is concerned, it can be stated that the outlook is much brighter, for though the infarctions show their greatest extent on the internal surface of the heart, the very rich and profuse subendocardial anastomoses generally supply sufficient nourishment to the superimposed Purkinje fibers to keep them intact.

Of course, where scarring has been so extensive as to involve the whole thickness of the wall and include the endocardium, there, undoubtedly, disappearance of the Purkinje fibers takes place, for it is very doubtful whether the ventricular blood can supply sufficient nourishment to keep the sensitive neuromuscular tissue alive. This does not, however, express itself necessarily in arrhythmias, since there is adequate and ample interanastomosing of the neuromuscular tissue within the chambers to supply and make up for gaps. On the other hand, it will be shown in Chapter VIII that the nutrition of the innermost layer of heart wall varies considerably in its supply with the age of the individual, becoming much

richer and better able to stand arterial obliteration as age progresses.

This has apparently been recognized to a certain extent by Haas, for in a case which he cites, where there was an atheromatous obliteration of the ramus septi ventriculorum superior with anemic infarction of the posterior portion of the septum and adjoining part of the left ventricle, the subendocardial musculature was intact. This, he explains, was due either to plentiful blood supply to this layer, or to nourishment from the blood contents of the ventricle.

Finally, Moenckeberg had already concluded that variability must exist in the blood supply to the heart and consequently in the original source of blood to the neuromuscular structure, for he observed that blockage of the same part of a coronary artery produced, in different hearts, different results in the bundle. This conclusion is amply supported by the variations which are described in Chapter III.

CHAPTER V

THE BLOOD SUPPLY TO THE HEART VALVES AND
ITS RELATION TO THE INFLAMMATIONS OF
THE VALVES

WITH the possible exception of the chapter dealing with the existence of anastomoses in the heart, no part of cardiac vascular morphology has been the subject of so much controversy as that on the blood supply to the valves.

Apart from its anatomical interest, Köster's acceptance of the existence of blood-vessels in valves as a basis for his theory of embolic endocarditis has brought additional importance to this question and has precipitated two distinct schools of thought. One of these denies the existence of vessels in normal valves and holds with Rühle that endocarditis¹ is caused by adhesion of bacteria from the main blood-stream to the valves. The selection of the closing edge as the usual primary seat of the lesion is, according to this opinion, due to the fact that the valve is here exposed to the greatest mechanical compression during diastole and systole, in the case of semilunar and auriculoventricular valves respectively. This, perhaps, together with a greater phagocytic power of the cells in this locality, accounts for the great frequency of endocarditis at this site. The other school claims that blood-vessels normally exist in valves and that bacterial emboli lodge in the site of greatest constriction,

¹The endocarditis referred to in this chapter is the valvular variety.

namely, where the delicate capillary arborizations take place at the closing edge; here, therefore, a septic focus with inflammation, rapid involvement of the overlying endocardium and thrombosis occurs.

Luschka was the first to claim that auriculoventricular as well as semilunar valves were vascularized. He stated that the semilunar valves receive their blood supply from the vasa vasorum as well as from the richly vascularized endocardium and that the auriculoventricular valves receive their blood supply from the attached edge as well as from the papillary muscles, through vessels which run along the chordae tendineae. He described these vessels as forming an arterial network in the main portion of the valve leaflets, accompanied by veins, and breaking up into more delicate strands which eventually end as capillaries at the closing edge. He also correlated the frequency of endocarditis on the aortic leaflet of the mitral valve with the relative ease with which blood-vessels can be found in this situation.

Rokitansky, Virchow, Joseph, and later Cadiat opposed these views, whereas Gerlach, Forster, Kölliker and Rosenstein confirmed them.

A third group, represented chiefly by Sappey, Frey, Henle and Coën arose, who stated that blood-vessels exist in the auriculoventricular valves, but not in the semilunar cusps.

Langer modified this view in pointing out that blood-vessels exist in those valves where muscle fiber remains are to be found.

This was followed a year later by Darier whose conclusions are in part as follows:

(a) There never exist any vessels in the fibro-elastic part of the auriculoventricular valves.

(b) The valves of the tricuspid and the left mitral leaflet are, as a rule, entirely fibro-elastic; the aortic leaflet of the mitral valve presents vasculature only in its upper part, generally not more than one-sixth of its length.

(c) In newborn children, one sees muscular bundles penetrating more or less to the fore in all auriculoventricular valves.

(d) In pathological changes in the valves, vessels are seen throughout the entire extent of the semilunar, tricuspid and mitral valves.

(e) These vessels seem to be the result of inflammations.

(f) Those writers who have succeeded in injecting the vascular network of the aortic and mitral valves have obtained pathological appearances.

(g) The valvular hematomata found in the newborn appear coincident with the processes of regression of vessels which occur in the valves during the fetal period.

Darier's conclusions have here been stated somewhat in detail, for in the subsequent discussion they acquire importance.

Odinzow later confirmed the results of Langer and Darier and these were again accepted by Königer.

Recently, Nussbaum has described a tortuous vasculature, which extends about 3 mm. from the base of the auriculoventricular valves into the leaflet, but no farther. He was unable to demonstrate blood-vessels in the semilunar valves.

In 1917 Bayne-Jones injected 14 human hearts: 6 from the first decade of life, 2 from the second, 2 from the third and 4 between the ages of thirty and sixty.

Out of these 14 hearts he was able to obtain a fairly complete injection in 3. In some of the others only irregular groups of vessels were seen which branched into delicate arterioles extending to the line of closure of the valves.

A description based on the three most completely injected hearts shows that the mitral and tricuspid valves receive a distinct vascularization from delicate twigs which arise from the annular branches of the right and left coronary arteries, and not

from vessels extending from the auricular musculature. The blood-vessels extend to the line of closure, where they form abundant anastomoses. Only occasionally do small vessels pass from the line of closure to the free edge of the valve.

In the upper portions of the valves Bayne-Jones was able to demonstrate veins which showed characteristic differences from the arteries.

The chordae tendineae showed blood-vessels which reached almost to the insertion into the valves, but never, in the human, actually passed on to the valve.

Bayne-Jones also succeeded in injecting 3 aortic and more pulmonary valves. The source of blood supply to these corresponds to the description already given by Luschka. The injection extended usually from the base of the cusp half-way to the edge. A few tiny vessels could be seen at the line of closure.

With all this conflicting evidence, therefore, it is not strange that there should be much dissension, and that some should claim, as does Darier, that blood-vessels exist only where there has been inflammation, and others, that blood-vessels occur in perfectly normal valves and that the idea of their non-existence is due to the inability of the observers to demonstrate them. Bayne-Jones, in fact, believes that technical difficulties account for most of the failures. He insists that the heart must be kept until rigor mortis passes off; that all cut edges as well as venous exits must be tied off; and that injection with a fine gelatine must be carried on at a pressure of from 160–190 mm. Hg to obtain satisfactory results.

It can easily be seen that the conception of the existence of blood-vessels in valves is essential to the theory of embolic endocarditis.

Finally, there is the third group to consider, represented

by Langer and others, who claim that blood-vessels occur in those situations where muscular remains persist.

From the experiments carried on by the author, as well as from a different interpretation of the literature on this question, it seems that each of these conflicting views is correct if taken in part, and that they can all be coordinated into a logical and reasonable explanation for the genesis and mechanism of at least many cases of acute valvular endocarditis.

As Tandler states, since Kürschner's description of the musculature found in the valves of human hearts, numerous investigators have followed with descriptions and studies of the frequency of occurrence of this tissue in the valves, of its relation to the age of the individual, and of its source from auricle or ventricle.

It seems quite clear that the auricular musculature which is quite abundant in fetal valves, becomes less so as the individual grows older. In children, as Langer, Darier, Manzone and Odinzow have shown, the musculature is still relatively abundant, but with beginning adolescence even this disappears and eventually, with few exceptions, the valve is rendered muscle-free. But these exceptions are of prime interest. One finds in these, bundles and columns of musculature which, in some cases, are relatively abundant and still maintain their original connection with the auricular musculature; in others, exist as isolated delicate strands.

The ventricular musculature is apparently much less important in this connection.

Langer's painstaking and accurate observations as to the coexistence of blood-vessels with this musculature is supported by numerous investigators. The fact that blood-vessels are found most easily towards the base of the valve

where the main mass of musculature exists, has been amply corroborated recently by Nussbaum and by the author.

The blood-vessels, moreover, become obliterated, lose their continuity with their main source (vessels which arise from the ramus circumflexus sinister and dexter as well as from the superior rami interventriculares), become blood-containing spaces (which perhaps accounts for the valvular hematomata so frequently found at birth) and eventually disappear.

This regression of blood-vessels proceeds hand in hand with the regression of the musculature; in fact, as Odinzow has pointed out, the musculature may entirely disappear before the vessels. In children where the muscular regression is incomplete, blood-vessels frequently persist.

If the literature on the mechanism of acute valvular endocarditis is now reviewed in this light, one is at once struck with the fact that the occurrence of endocarditis bears a strikingly close relationship to that of the existence of musculature and of blood-vessels in valves. This becomes even more striking when the incidence of endocarditis is considered from the point of view of the frequency with which it occurs on the left and right sides as well as on the individual valves and cusps.

In the fetus, where the valves, in keeping with the musculature, are undoubtedly better vascularized on the right side of the heart, endocarditis is much more frequently found on the right side than on the left.

In children, where the complete regression of vasculature has not yet occurred, endocarditis is relatively frequent.

Finally, in adults, the most frequent seat of endocarditis is the aortic cusp of the mitral valve, the very leaflet which

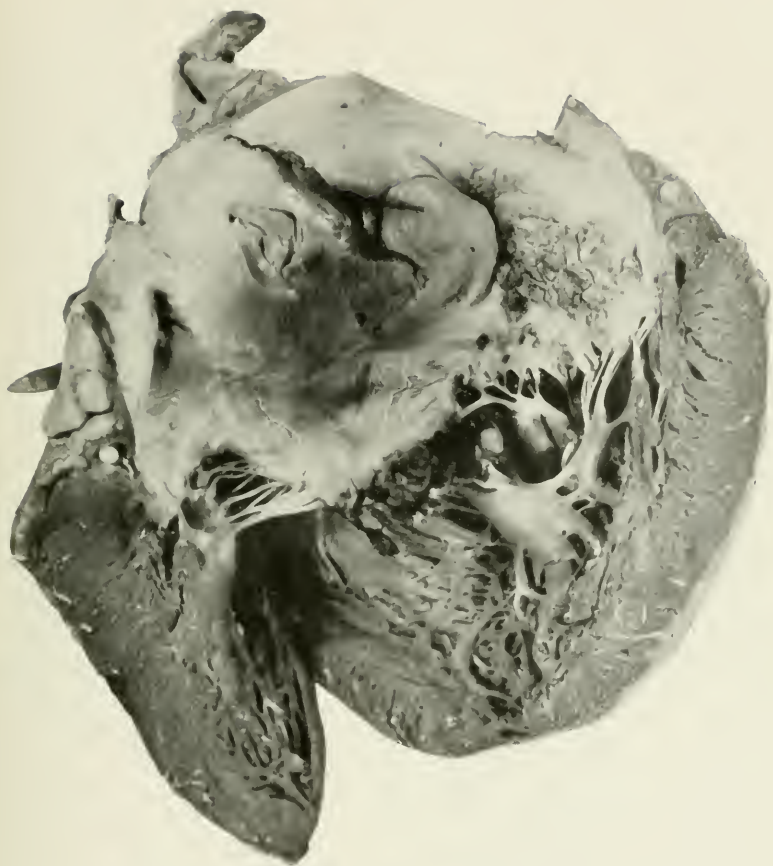


FIG. 9.—Photograph of injected specimen, showing the blood supply to the aortic cusp of the mitral valve which is the seat of an acute endocarditis.



FIG. 10.—Photograph of the tricuspid valve from the same heart as in Fig. 9.



FIG. 11.—Photograph of the pulmonary valve from the same heart as in Fig. 9.



FIG. 12. — Photograph of the aortic valve from the same heart as in Fig. 9, showing the blood supply to the cusps.

Langer has already shown to be the last to lose its musculature, and which, as so many observers have found and the author's injections have entirely corroborated, is the most usual site of complete injection.

That practically all cases of acute valvular endocarditis show a well-developed vasculature seems certain. The author has not yet failed to demonstrate it in such cases. These vessels cannot be, as Darier claimed, entirely secondary to the inflammation, for they are quite orderly in arrangement, possess veins and bear no resemblance to granulation tissue. Moreover, their capillary terminations extend to the closing edge of the valve when this is the seat of the lesion; and it is rather forced to assume that granulation tissue extends through the whole of so large a valve as the aortic leaflet of the mitral, to the terminal edge in order to supply the seat of bacterial adhesions with a well-organized vasculature.

Figure 9 shows a typical injection from such a case. It will be seen that several delicate vessels descend from the insertion of the aortic cusp of the mitral valve at its base. The most anterior of these twigs arise from the first portion of the ramus circumflexus sinister; the posterior branches, from the uppermost rami interventriculares. Descending to about the middle of the leaflet, they anastomose in the form of an arch, from the convex border of which numerous interlacing and interanastomosing arborizations are given off. (This is even better seen in the cleared specimen.) Towards the closing edge of the valve, where the fungoid vegetative lesion is seen, a final capillary breaking-up occurs to supply richly this site.

The posterior cusp of the mitral valve shows no vascularization but is the seat of apparently more recent thrombotic vegetations which extend up the wall of the left auricle.

The tricuspid (Fig. 10) as well as the pulmonary valve (Fig. 11) are free from endocarditis and show no vasculature. The aortic valve, however, shows in all three cusps, vessels which extend from the base to the middle (Fig. 12). In this connection, it is very interesting to find that, in this specimen, the aortic valve, which is so frequently the seat of an endocarditis following a mitral lesion, should show a vasculature, and one wonders whether, if this patient had lived long enough, an endocarditis would not have occurred here.

Normal valves can certainly be injected, but only exceptionally. The author has succeeded in injecting the mitral valve in about 6 per cent of normal hearts which, however, represented all age periods from birth to the ninth decade of life. Figure 13 shows such an injection; in this case only the aortic leaflet of the mitral valve showed vasculature. The course of the vessels is very much the same as that found in the case of endocarditis.

When a complete injection of all the valves is obtained, it is found that the posterior cusp of the mitral receives its blood from the terminal portions of the ramus circumflexus sinister and dexter, and that the tricuspid valve receives its blood from the ramus circumflexus dexter as well as from the superior rami interventriculares, according to the position of the leaflets. Where the ramus circumflexus sinister extends beyond the crux, the posterior leaflets of the mitral and tricuspid valves receive their blood from this vessel.

The frequency with which these valves can be injected depends upon the age of the individual. It will be observed that out of the 14 carefully selected human hearts which Bayne-Jones injected, 10 belonged to the first thirty years of life, and of these again 6 were from the first decade; in spite of

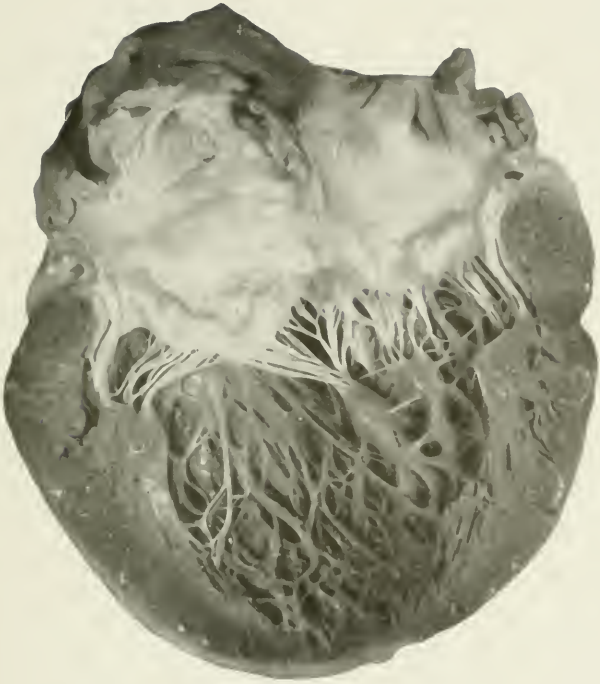


FIG. 13. — Photograph of injected specimen, showing the blood supply to the aortic leaflet of a normal mitral valve.

this, he was able to inject fairly completely, only 3 cases. It is the author's belief that the other cases could not be injected because vessels did not exist. In fact, the frequency with which he succeeded in these cases is due to the fact that in young hearts, where muscular remains still occur fairly frequently, the vasculature is persistent in the same ratio.

In the hearts which the author injected, every possible precaution was taken to eliminate artefact and to ensure a perfectly standardized injection. Each heart was subjected identically to the same procedure as described in Chapter I. In spite of this, only about 6 per cent of the hearts showed valvular injection, and of these, the aortic cusp of the mitral valve was the most frequent seat.

Dr. M. Notkin examined in our laboratories microscopically and in serial section the valves which showed *no* injection and was unable to demonstrate in them any blood-vessels or capillaries, thus substantiating the conclusion that these valves showed no injection because vessels were non-existent.

If the foregoing facts are now arranged in orderly form, one may begin with the following premises:

1. Fetal valves contain musculature on right and left sides, the right probably being richer in blood-vessels.
2. Fetal valvular endocarditis is found more frequently on the right side.
3. Regression of musculature and blood-vessels occurs as age advances, but infant's valves still frequently contain both.
4. Infants are frequently attacked by valvular endocarditis.
5. In adults, valvular endocarditis is not as frequent as in children but occurs with preference on the aortic cusp of the mitral valve.
6. The aortic cusp of the mitral valve is the last to show

regression of musculature and the most frequently injected leaf.

7. Practically all cases of valvular endocarditis show a distinct vasculature.

8. Relatively few normal heart valves show in the adult a vasculature, the frequency being somewhat greater than the occurrence of persistent muscular strands.

To these may be added:

9. Hearts which show congenital anomalies, arrests in development, very frequently show also an endocarditis, and this, moreover, is more frequently the case on that side and at that location where the arrest of development is seen, particularly where this bears a close association with interference with the embryonic valve *anlage*. In such cases one would expect perversions and arrests of normal vascular regression on the same side.

This view is lent considerable support by a study, very kindly made for the author by Dr. Maude E. Abbott, on the incidence of acute endocarditis in 581 congenital cardiac defects. The following is a short excerpt in table form from these cases:

| | Number of Cases | Acute Endocarditis |
|---|--------------------|-----------------------|
| <i>Anomalous Septa</i> | | |
| In left auricle. | 7 | 0 |
| In right auricle... | 3 | 1 |
| In ventricles..... | 2 | 0 |
| <i>Defects of Auricular Septum</i> | | |
| Patent foramen ovale..... | 19 | 0 |
| High auricular septal defect. | 9 | 0 |
| Low auricular septal defect..... | 12 | 4 |
| <i>Defects of Interventricular Septum</i> | | |
| Defects at base..... | 34 | 9 |
| Defects elsewhere than at base. | 3 | 0 |

(The four cases of acute endocarditis occurring in the low auricular septal defects with persistent ostium primum and consequent cleavage of the anterior segment of the mitral valve, presented vegetations on the valves of the left side.) In this group, the close relationship between the occurrence of acute endocarditis and defects which involve the region of the auriculoventricular valve *anlage*, is of considerable significance.

| | Number of Cases | Acute Endocarditis |
|---|--------------------|-----------------------|
| <i>Cor Biloculare and Triloculare</i> | 23 | 0 |
| <i>Defect of Aortic Septum</i> | | |
| Complete defect (persistent truncus) | 13 | 0 |
| Partial defect (with defect of ventricular septum) | 1 | 1 |

Here it is found that a complete absence of septum formation is not associated with the occurrence of acute endocarditis. In the cases, however, where there was an aborted attempt at this formation, endocarditis was found.

| | Number of Cases | Acute Endocarditis |
|--|--------------------|-----------------------|
| <i>Pulmonary Stenosis</i> | 82 | 21 |
| <i>Pulmonary Atresia</i> | 24 | 1 |
| <i>Subaortic Stenosis</i> | 8 | 3 (on left side) |
| <i>Aortic Stenosis</i> | 6 | 0 |
| <i>Aortic Atresia</i> | 3 | 0 |
| <i>Tricuspid Stenosis</i> | 2 | 0 |
| <i>Tricuspid Atresia</i> | 10 | 2 |
| <i>Mitral Stenosis</i> | 1 | 0 |
| <i>Mitral Atresia</i> | 3 | 0 |
| <i>Anomalies of Auriculoventricular Valves</i> | 12 | 0 |

Although not so suggestive, these figures indicate the importance of defects which produce stenosis, in connection with endocarditis, and emphasize the frequency with which this occurrence is on the right side.

Other cases are considered which, though not so pertinent to this question, indicate, nevertheless, that the anatomical factor of narrowing vasculature, which may indeed be second-

ary to a wear-and-tear scarring process, is intimately associated with the occurrence of endocarditis.

It would appear from these premises that only a certain percentage of postnatal hearts contain persistent fetal vasculature of valves and that valvular endocarditis may be caused by embolization of bacteria in these persistent vessels. But as this persistence is, fortunately, relatively infrequent, it follows that the incidence of valvular endocarditis, resulting from an embolizing bacteriemia due to organisms which are causative agents of this disease, will bear a relation to the frequency with which blood-vessels are found in the valves. It would, therefore, be important to obtain figures of this incidence at different age periods in relation to the occurrence of blood-vessels in the valves. This figure, it is believed, would probably represent very closely the proportion of cases of endocarditis.

Just as an individual in whom other fetal structures persist, is exposed to the possibility of their disease, so, it is the author's opinion, the individual, whose valvular vasculature has not undergone regression, is liable or predisposed to a valvular endocarditis.

If endocarditis were caused only by bacterial adhesions to the closing edge of a valve where compression is greatest, it does not seem quite clear why the aortic cusp of the mitral valve should be the most frequent to suffer. Nevertheless, it is not the author's intention to deny the possibility of valvular infection by way of the main blood-stream, as indeed it seems to occur in parietal endocarditis (toxic form as shown by de Vecchi, or bacterial form, by Ribbert), but it appears desirable to emphasize that in view of the considerations presented above, it is probable that an individual who has

persistent valvular blood-vessels is certainly additionally exposed to endocarditis. Here the compression at the edges would serve as still another factor, besides the narrowing of the blood-stream, for precipitation of bacterial clumps.

The heart valves are peculiar in that they are the only structures in the body which undergo periodic compressions by a force equal to the weight of approximately 100 mm. Hg and over. Moreover, these compressions, according to Howell, occur seventy times a minute and last 0.379 second in the case of auriculoventricular valves, and 0.483 second in the case of semilunar valves. In other words, the capillaries in the edges of the auriculoventricular valves are compressed for 10.612 hours during the day, and those of the semilunar valves, 13.524 hours. This would afford an excellent opportunity for the arrest and development of pathogenic bacteria.

It may be argued that the incidence of endocarditis in certain infections, such as rheumatic fever, is so common that the relative infrequency of persistent vasculature in valves is here of little importance. But this objection loses much weight in the light of stricter criticism, for hospital statistics are no indication of the relative proportion of the occurrence of endocarditis in rheumatic fever. Moreover, Osler's statement and general clinical experience, that the insusceptibility to endocarditis diminishes as age advances, are strongly suggestive of the influence of age on the regressive anatomical changes in the valvular vasculature.

It must also be borne in mind that other factors besides the mechanical may enter into the occurrence of an endocarditis; for, whereas certain bacteria may require compression for settlement and action at the closing edge of a valve, other, perhaps more virulent, organisms may be enabled

to localize and set up inflammation in a part of a vessel not submitted to this mechanism. Thus, ulcerative endocarditis is known to arise relatively frequently in other portions of the endocardium and in the valve leaflet. Here, the fact that occasionally the valvular vessels terminate before reaching the edge, may serve as a focus of bacterial arrest. This was well illustrated in several typical cases recently injected by the author.

Finally, through lack of sufficient material the author unfortunately cannot give accurate statistics from his own observations on the frequency with which valves can be injected during the different age periods, but it would be of great importance to obtain exact information in regard to the percentage of successful injections in increasing age, for it appears probable that the latter would be accompanied by a decreasing percentage.

Those who have attempted to inject blood-vessels in valves have, therefore, failed in a great many instances, not only on account of an imperfect technique, but doubtless also because of the normal evolution of heart valves which in adult life usually causes a complete disappearance of vessels.

CHAPTER VI

THE ANASTOMOSES BETWEEN THE CORONARY ARTERIES

EVEN though the question as to the existence of anastomoses between the coronary arteries has been the subject of long controversy and discussion, it can now with certainty be stated that, to this at least, a final answer has been given.

The question of anastomoses resolves itself into three groups:

(1) Do anastomoses exist between the right and left coronary arteries both in their capillary and precapillary distribution?

(2) Do anastomoses exist between branches of each coronary artery?

(3) Do anastomoses exist between the coronary arteries and vessels of the adjacent and attached organs?

The sources of information on these questions have been extraordinary both in the number of contributors and investigators, as well as in the methods employed for its elucidation.

The following classification shows how varied were the factors which the different observers employed singly and in combination in these investigations:

(a) Dissection.

(b) Experimental tying-off of coronary arteries or their branches, chiefly in dogs, to determine, by evidence of infarct formation or rapidity with which heart stoppage occurred, the existence or non-existence of anastomoses.

- (c) Injection of colored gelatines into both arteries.
- (d) Injections of a colored gelatine into one coronary artery to see whether it passes out of the other.
- (e) Serial section and reconstruction of injected specimens.
- (f) Injection of metals with subsequent corrosion.
- (g) Injection of gelatine suspensions of heavy salts with subsequent roentgenography.
- (h) Injection of colored gelatines with clearing of the remaining tissue.
- (i) Examination and dissection of pathological specimens showing obliteration of branches.
- (j) Observation of clinical cases with, frequently, autopsy examinations.

To these, the author has added:

(k) Injection of vessels with a gelatine suspension of a heavy salt by a special technique which standardizes all mechanical factors. This is followed by stereoscopic roentgenography of the organ, clearing, dissection and microscopic section. Normal as well as pathological hearts were used for the purpose. (See Chapter I.)

The first historical experiments on the effect of tying-off a coronary artery in a dog were those made by Chirac in 1698. These, however, threw no light on the question of anastomoses, since the only observation made by him was that the heart ceased beating.

It was not until 1708 that Thebesius, followed later by Haller, Morgagni and Sénac, on the basis of careful dissections, came to the conclusion that anastomoses exist between both coronary arteries. Haller stated that these were quite rich and occurred with frequency at the root of the pulmonary artery, in the posterior sulcus longitudinalis, in the right ventricle,

at the apex of the heart, on the surface of the ventricles and through the vasa vasorum of the great vessels.

In 1799 Parry and Jenner (cited by Parry) first interpreted the clinical syndrome known as angina pectoris as due to calcification of the coronary arteries and the autopsy findings on John Hunter's heart, after Jenner had diagnosed his condition, corroborated this view.

In 1810 Caldani's dissections revived the claim that anastomoses exist, particularly at the root of the pulmonary artery. Cruveilhier again described wide anastomoses between both coronary arteries as well as with bronchial arteries.

In 1842 Erichsen published his results of experimental ligation of coronaries in animals and concluded that "any circumstance that may interfere with passage of blood through the coronary arteries either directly, as in ossification of the coats of those vessels, or indirectly, by there not being sufficient blood sent out of the left ventricle as in cases of extreme obstruction or regurgitant disease of the aortic or mitral valve, may occasion the fatal event."

In 1855 Hyrtl, on the basis of injection and corrosion experiments, categorically denied the existence of anastomoses between the coronary arteries and this was confirmed in 1866 by Henle, who stated, however, that capillary anastomoses do occur.

Krause was the first to oppose the views of Hyrtl, but meanwhile Béraud had found that anastomoses exist between the coronary arteries and vessels from adjoining organs.

Panum, von Bezold and Breymann and later Samuelson again experimented with dogs and were able to confirm Erichsen's conclusions.

In 1880 Langer showed that anastomoses exist between

the coronary arteries and those of the pericardium and, through these, with the arteriae mammae internae. He showed further that by means of the vasa vasorum of the pulmonary artery, connection also takes place with the bronchial arteries and, through branches from the auricles, with the diaphragm.

In 1881 Cohnheim and A. von Schulthess-Rechberg reported their experiments on the clamping of coronary arteries in curarized dogs. Their conclusions, which profoundly influenced the opinion of future observers and which are still being held by some, were, that clamping of either main coronary artery caused the ventricles to stop in diastole within two minutes. They accordingly argued that the coronaries were end-arteries, and that, if any anastomoses exist, they must consist of fine capillaries.

This was later confirmed by G. Sée, Bochefontaine and Roussy, Bettelheim and Kronecker. The ligature experiments thus far made, together with injections carried out by Dragneff, Zimmerl and Banchi which again confirmed Hyrtl's work, helped to lend much support to the opposers of the view that anastomoses exist.

It was not long, however, before a great many observers, notably McWilliam, Fenoglio and Drogue, Bickel, Kölster, Tigerstedt, von Frey and Porter, after performing very carefully numerous ligation experiments in dogs, came to conclusions opposed to those of Cohnheim and von Schulthess-Rechberg. In general, they held that many of the latter's results were due to the trauma of the operation, and that tying-off branches and even a main coronary artery does not necessarily lead to instantaneous death. In 1892 Kölster gave an accurate description of the processes of infarct formation and healing by scarring.

This opposition, moreover, further gained strength by the collection and description of numerous clinical cases and pathological material by Samuelson, Huber, Aschoff and Tawara, Huchard, West, Chiari, Pagenstecher, Engelhardt, Thorel, Dock, Galli, Merkel, Osler, Krehl and, recently, Herrick. These observers were able to show that in the human heart, obliteration of coronary branches, and in some cases a main coronary artery, produced results which varied in the different cases from almost instantaneous death to those which experienced no symptoms and showed absolutely no clinical sign, the condition being recognized only at post-mortem examination following death from some intercurrent disease. Some of these observers, too, were able to make out by dissections distinct anastomoses.

It now remained to describe exactly the location and appearance of these anastomoses and to explain why, if anastomoses exist, infarcts occur.

In 1907 Jamin and Merkel elaborated and improved on Freyett's method of radiographing injected coronary arteries and presented a stereoscopic radiographic study of 29 hearts whose coronary arteries were injected with a 10 to 15 per cent suspension of red lead in gelatine. They concluded that great individual differences exist in the anastomoses and that these are found most frequently in the auricle, interauricular and interventricular septum and, in special instances, on the anterior wall of the right ventricle, over the papillary muscles and the apex of the heart. In pathological specimens, the anastomoses were found especially in the interventricular septum and the anterior wall of the left ventricle.

In the same year Spalteholz employed a chrome-yellow suspension in gelatine for injections with subsequent dehydra-

tion and clearing in benzol and carbon disulphide. By this method he was able to obtain a reconstruction of the cardiac circulation which, on the whole, was vastly superior to anything hitherto obtained.

Spalteholz's conclusions are as follows:

- (a) No end-arteries exist in the heart.
- (b) Rich anastomoses occur in all layers of the heart and, through the vasa vasorum, on the great vessels.
- (c) In the thick muscle of the left ventricle, perpendicular vessels penetrate to anastomose under the endocardium.
- (d) The papillary muscles are particularly rich in anastomoses.
- (e) With growth, the appearance of vessels on the surface show a typical alteration.

Hirsch performed a series of experiments on dogs in which he tied off the ramus descendens anterior and observed infarct formation. In all normal cases, however, he found that the infarcted region was much smaller than the area of supply of the tied-off vessel. In one instance where the animal had previously lost much blood, the infarcted area corresponded with the entire musculature supplied by the vessel. A similar observation had already been made on the human heart by von Recklinghausen and Fujinami.

In 1909 Miller and Matthews were able to prove that many of the fatal results obtained by Cohnheim, Fenoglio and Drogue, Porter, etc., were due to the usage of curare and morphia. By using ether as an anesthetic and employing strophanthus as a heart tonic, they obtained a mortality of only 8.7 per cent after ligation of the ramus descendens anterior of the left coronary artery. Even after tying off a main branch of the ramus descendens anterior, the animal would recover for a period varying from one to three months and ultimately die of acute cardiac decompensation. They

were, therefore, of the opinion that considerable anastomoses exist between both coronary arteries.

In 1910 Amenomiya made a study of the blood supply to the papillary muscle and found only capillary anastomoses.

In 1911 Nussbaum described direct connections between arteries and veins, made up of a single layer of endothelium possessing no muscular coats and lying in the subendocardium. He considered these as safety outlets for arterial blood when the pressure becomes too high.

In Herrick's clinical classification of angina pectoris (1912) one group of cases concerns patients who survived an obliteration of a coronary artery for a period of time which varied from days to weeks. All these occurred in individuals over fifty years of age. He was of the opinion that anastomoses exist and that the condition of the heart musculature and patency of the vessels played an important part in determining the degree of compensation which can take place after obliteration. Gradual obliteration, he argued, allows the heart to adapt itself to the new conditions and allows collaterals to develop sufficiently to compensate. He suggested that the vessels of Thebesius might serve as accessory nutritive channels in such cases.

Finally, very recently Smith made an experimental study on the question as to the existence of anastomoses. By observation on dogs, as well as interpretations of human material, he concluded for the following reasons that anastomoses exist:

- (a) Survival of dogs even after tying-off a relatively large vessel.
- (b) Variability of the lesion.
- (c) Relatively small size of the lesion.

Thus, it cannot any longer be doubted that anastomoses exist between the branches of an individual coronary artery as

well as between branches from both sides. The dissections of Thebesius, Haller, Morgagni, de Sénac and Caldani, the experimental work by McWilliam, Fenoglio and Droguell, Bickel, Kölster, Samuelson, Tigerstedt, von Frey, Porter, Miller and Matthews and Smith; the clinical and anatomical observations made by Samuelson, Huber, Aschoff and Tawara, Huchard, West, Chiari, Pagenstecher, Engelhardt, Thorel, Dock, Galli, Merkel, Osler, Krehl and Herriek, as well as the injection work of Jamin and Merkel, Spalteholz and Nussbaum have placed this conclusion beyond dispute.

There is still, however, no accurate knowledge of the exact nature as well as architectural arrangement of these anastomoses. The author has accordingly made a very careful study of this question by the methods described in the chapter on technique and has come to the general conclusion that the heart is perhaps the richest organ in the body as regards capillary and precapillary anastomoses between branches of the same coronary artery as well as between branches from both coronaries. The detailed architectural description has been left to this chapter since it represents a category in itself.

Figure 24 shows a photograph of an injected and cleared specimen which illustrates beyond any dispute the abundant network of anastomoses at the root of the pulmonary artery. A constant arterial arch is found crossing the first part of the root. It corresponds to the venous arch seen in this locality. The first part of the aorta shows similarly extensive anastomoses between the vasa vasorum which arise from each coronary artery.

On the surface of the heart (Figs. 14 and 14 A) a good injection will show a very open anastomosis occurring between branches from the ramus descendens anterior of the left coro-

nary artery and those from the rami anteriores of the right. A similar, though usually less conspicuous, anastomosis occurs on the corresponding posterior surface between the rami marginis obtusi and the rami ventriculares sinistri posteriores of the right coronary artery.

The interauricular and particularly the interventricular septum is the seat of very extensive and, in certain age periods (Chapter VIII), very wide anastomoses.

So far as communication between smaller vessels of the heart is concerned, the auricular walls and appendages, as well as the ventricular walls throughout, are the seat of very abundant anastomoses and interanastomoses between branches from both coronaries as well as between branches from each coronary artery. To this may also be added the anastomoses which occur between the vessels which supply (when this occurs) the valve leaflets (Fig. 13).

Figure 5 shows the very complex and complete anastomosis of small vessels, which takes place beneath the endocardium of the ventricular walls and papillar muscles and within the musculature of the latter.

Capillary anastomoses are very numerous and rich and can be seen in any portion of cardiac musculature.

Finally, an important factor in anastomoses and one which, as will be seen in Chapter VIII, assumes, particularly in the later age periods, considerable functional significance, is that which occurs between the arteriae tela adiposae cordis, those vessels which lie in the fatty tissue under the visceral pericardium. The author has traced these vessels into the auricular as well as ventricular musculature and has found distinct anastomoses between these and branches from the coronary arteries and vasa vasorum.

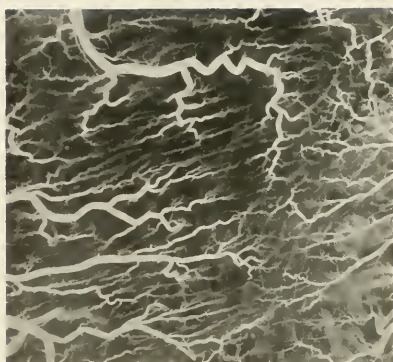


FIG. 14 A.—An enlargement from Fig. 14.



FIG. 14.—Photograph of injected and cleared specimen, showing the anastomoses on the anterior surface of the heart.

Lastly, the author has found distinct connections between the coronary arteries and the vessels in the parietal pericardium, so that this, together with Langer's observations as to the existence of anastomoses between the coronaries and branches of the bronchial arteries, arteriae mammae internae and those of the diaphragm, render beyond dispute the fact that distinct connections exist between the cardiac vasculature and that of the adjacent organs.

If one now turns back to the first questions with which the discussion on this chapter was opened, it is found that the following statements can safely be made:

(a) Anastomoses exist between the right and left coronary arteries both in their capillary as well as precapillary distribution.

(b) Anastomoses exist between the branches of each coronary artery.

(c) Anastomoses exist between the coronary arteries and vessels from the adjacent and attached organs.

(d) Anastomoses in the heart are universal and abundant.

The question which now arises is how to explain, on this basis, the formation of infarcts, for it has been shown that end-arteries, in the anatomical sense of Cohnheim, do not exist in the heart.

Pratt has formulated a definition in which he includes, in the category of *functional end-arteries*, that vascular structure in which the resistance in the anastomotic area is greater than the pressure in the different vessels. To a certain extent only do the anastomoses of the heart fall under this definition.

It has been stated that Hirsch found in his experimental work on dogs, and this has been confirmed by von Recklinghausen and Fujinami as true for the human heart, that in normal

hearts the infarcted area is smaller than that supplied by the obliterated vessel. When, however, the pressure in the blood is lowered or perhaps when the blood is rendered poorer in its composition, the infarct corresponds completely to the area supplied by the vessel. This occurred in one dog upon which Hirsch experimented.

Hirsch concluded from these experiments that infarcts can occur in the heart, despite the anastomoses, because the heart is always functioning. He believes further, that the direction and extent of the anastomoses, the structure of the vessels, the heart strength and the time and duration of the obliteration are important factors in this connection.

Amenomiya concluded from his study of infarcts occurring in papillary muscles that for an infarct to occur it is necessary to have:

- (a) Too little anastomoses.
- (b) Closure of relatively large vessels.
- (c) Rapid blockage of the vessel.

It is generally the case that the infarcted area is situated on the inner aspect of the heart (Smith, Oppenheimer and Rothschild), but it is occasionally found that the infarct occurs in the outer side of the wall, leaving the inner surface intact. This variability suggests that either other factors besides the anatomical construction can dictate the occurrence or non-occurrence of infarcts, or that the anatomical structure is fluid, or both.

Spalteholz, Galli and the author (Chapter VIII) have found cases where complete and almost complete obliteration of a coronary artery have produced no lesion in the myocardium.

The age of the individual is of prime importance in this connection, for as will later be shown, the older the individual,

the more free and patent are the anastomoses. An old heart, is therefore much more prepared to receive with relatively little or no damage, the brunt of a sudden obliteration of a nutrient vessel.

Moreover, as Herrick has stated, a gradual obliteration of a vessel allows time for the existing anastomoses to widen and compensations to take place. Here the arteriae telae adiposae are of considerable importance, more especially in the heart of older individuals.

The occurrence of an infarct on the outer rather than on the inner aspect of the heart, as in a case shown to the author by Dr. Rothschild, was undoubtedly due to the fact that here the subendocardial anastomoses were rich enough to supply the anemic area.

If the foregoing facts, therefore, are summed up, it can be concluded that in the ordinary course of events and in the average young adult's heart, the intricate systems of anastomoses are all in active function and are not prepared to act suddenly as entirely adequate compensatory agents. Nevertheless, when vascular obliteration takes place, a certain amount of compensation does occur, so that the infarcted area is smaller than the region supplied by the obliterated vessel, the remaining portion receiving sufficient nutrition from the anastomoses. Moreover, if the obliteration is gradual and the circulation good, sufficient dilatation of the anastomosing vessels can occur to preserve considerable, if not all, of the musculature. When the obliteration occurs in a relatively older individual's heart, the patent and free anastomoses, as well as the well-developed arteriae telae adiposae, can often amply supply the affected area so that the myocardium can be completely spared.

Thus, it is seen that in the determination of infarct formation, besides the factors of size of the obliterated vessel, its location, the duration and rapidity of the obliteration, the condition of the general circulation and that of the heart musculature, another very important one must be added—namely, the age of the individual.

CHAPTER VII

THE VEINS OF THE HEART

ON account of their rather complicated embryogenetic development, the veins of the heart are prone to considerable variation.

As in the case of the arteries, a description will here be given of the venous structure in the average heart (Fig. 15) and this will be followed by a short discussion on the variations. To render the latter somewhat more comprehensible, a brief description of the embryogenesis of the heart veins will precede the description of the variations.

The veins of the heart can be divided conveniently into:

- (A) *Venae magnae cordis.*
- (B) *Venae parvae cordis.*
- (C) *Venae minimae Thebesii.*

A. *VENAE MAGNAE CORDIS*

The term *Venae magnae cordis* has been chosen as a convenient category for the largest veins of the heart. This must not be confused with the vessel which encircles the left auriculoventricular furrow to empty into the sinus coronarius and which is called by some *vena magna cordis*. The author feels that the term *Vena coronaria sinistra* is much more suitable for the latter, and one less open to confusion.

Sinus coronarius. The first great vein which is to be considered is the *Sinus coronarius*. It really serves largely as a collecting receptacle for blood poured into it from the

remaining large veins of the heart, and which it in turn transmits to the right auricle.

There has been considerable dispute as to whether the term *Sinus coronarius* should include only that portion of the vein posteriorly which is covered by musculature (Reid, Marshall) or extend beyond this to limits defined by various observers (Portal, Winslow, Cruveilhier). As Tandler points out on the basis of Marshall's embryogenetic classification, the most satisfactory boundaries are the valvula Thebesii proximally, and the valvula Vieusenii distally. It is usually surrounded in its entire course by heart musculature.

The sinus coronarius thus delimited lies in the posterior auriculoventricular groove and extends, usually as a short thick trunk covered with transverse musculature, from a point about halfway between the margo obtusus and the crux, to a point just to the left of the crux.

Vena coronaria cordis sinistra. Opening directly into the sinus coronarius through the valvula vieusenii and continuous with it, is the *Vena coronaria cordis sinistra*. This is a large vein which tapers from its origin as the continuation of the vena interventricularis anterior at the junction of the auriculoventricular sulcus, becoming larger as it rounds the margo obtusus in the furrow to become eventually continuous with the sinus coronarius.

Vena interventricularis anterior. This vein commences usually at the lower third of the anterior interventricular sulcus in a wide anastomosis with the corresponding posterior vein. It ascends the sulcus in company with the ramus descendens anterior of the left coronary artery and, at the auriculoventricular sulcus, becomes continuous with the vena coronaria cordis sinistra.



FIG. 15.—Roentgenogram of the venous distribution in the average heart.

Vena coronaria dextra. Another constant vein which opens into the sinus coronarius, is the *Vena coronaria dextra*. This vein is rather slender, lies in the auriculoventricular furrow in the right posterior aspect of the heart and is often the direct continuation of the vena marginis acuti.

Vena marginis acuti. This smaller vein commences on the lower third of the margo acutus as a distinct anastomosis with one of the terminal branches of the vena interventricularis posterior. It rounds the junction of the margo acutus with the auriculoventricular sulcus to become the vena coronaria dextra. As will later be shown, it may also open independently into the right auricle.

Vena interventricularis posterior. This large and tapering vein commences in the lower third of the anterior interventricular sulcus. Anastomosing at its origin with the vena interventricularis anterior, and receiving anastomotic twigs from the vena marginis acuti and venae marginis obtusi, it rounds the apex and ascends the posterior interventricular sulcus to empty into the sinus coronarius.

Branches Which Enter the Venae Magnae Cordis

Vena obliqua atrii sinistri (Marshalli). Because it is of considerable embryogenetic interest and marks as well at its entrance the beginning of the sinus coronarius, the delicate auricular vein known as the *Vena obliqua atrii sinistri* merits first description. This vein commences on the anterior surface of the left auricle and, proceeding between the two left pulmonary veins, courses diagonally downward and toward the right of the heart to empty into the sinus coronarius about the site of the valvula vieusenii.

Venae ventriculi sinistri. These are very large veins which,

on the left side, carry blood centripetally from a point two-thirds down the margo obtusus as the center, to empty into the venous ring formed by the linking up of vena coronaria cordis sinistra, vena interventricularis anterior and vena interventricularis posterior.

According to their situation they are called *Venae ventriculi sinistri posteriores, marginales* or *anteriores*.

Venae ventriculi dextri. On the right side, the venous chain is not so large and consists of the linking up of vena marginis acuti, vena coronaria dextra and vena interventricularis posterior.

Into this ring there course centripetally and empty, veins which are not so large as those found on the left side. These are known on the posterior surface of the right ventricle as *Venae ventriculi dextri posteriores*. Here they usually pursue a remarkably even horizontal course, parallel one to the other.

Finally, in the interventricular septum there are found numerous, large and richly interanastomosing veins which empty into the anterior and posterior venae interventriculares.

The topmost of these empties into the vena interventricularis posterior. It accompanies the ramus septi fibrosi through the bundle and shows a wide anastomosis with a more delicate vessel which empties into the vena interventricularis anterior.

There remains the description of the veins draining the anterior surface of the right ventricle. This is accomplished by two systems: (1) *venae ventriculi dextri anteriores*; (2) *venae parvae cordis*.

The *Venae ventriculi dextri anteriores* are transverse richly anastomosing veins which empty into the vena marginis and vena interventricularis anterior.

B. VENAE PARVAE CORDIS

It will be seen that the auriculoventricular sulcus is surrounded by veins in its whole circumference with the exception of that part which lies between the margo acutus and anterior interventricular sulcus.

It is over this portion of the sulcus that the second group of veins, draining the anterior surface of the right ventricle, cross to empty directly into the right auricle.

These consist of three or four relatively small veins which run parallel to one another and show wide anastomoses in their ventricular aspect.

Two other *venae parvae cordis* deserve some special mention. One, which empties into the right auricle between the root of the pulmonary artery and the appendage, corresponds to the constant arterial arch already described at the junction of the conus with the pulmonary artery (Cruveilhier) and anastomoses with a corresponding vessel which empties into the right auricle after draining blood from the roots of the aorta and pulmonary artery and adjacent portion of the right auricle (Zuckerkindl).

For the rest, a number of small auricular veins which empty for the most part into the *vena coronaria sinistra* and which show some correspondence with the auricular arteries, constitute the remaining *venae parvae cordis*.

Of the veins thus far described, the main trunks usually accompany branches of the coronary arteries and, in these cases, occupy a position beside them or lying upon them. The smaller ramifications may lie subjacent to the arteries. The deeper divisions of the veins differ from the arteries in that they do not form so regular a series of dichotomous branching

but course to the surface as somewhat tortuous, delicate, interlinking channels which join up at the surface and abruptly empty into the larger superficial trunks of the second and third order. At the apex, there is often seen a well-formed medusa-head whorl of delicate veins which empty into the terminal branches of the *venae interventricularis anterior* and *posterior*. At the root of the aorta and in the subpericardial fat, a well formed structure of *venae telae adiposae* can also be formed.

C. VENAE MINIMAE THEBESII

It has been seen that with the exception of the *vena obliqua atrii sinistri* and the vein described by Zuckerkandl, there are practically no veins of larger caliber draining the auricles. This lack is largely made up for by the existence of tiny venous channels known as *Venae Thebesii* according to their discoverer Thebesius, who described them independently and unaware of the fact that Vieussens had already made known their presence.

Their existence has been open to much discussion, having been confirmed by Winslow, Verheyen, Lancisius, Bochdalek, Henle, Hyrtl and others, and denied by Sénac, Zinn, Haller, Cruveilhier, Theile and Luschka.

Bochdalek, moreover, proved their existence in both auricles and this was later confirmed by Langer. Those who denied the existence of these tiny channels as carriers of venous blood, held them to be blind diverticulæ. Haller claimed that their function as veins meant admixture of venous blood with the arterial on the left side, which seemed improbable on physiological grounds.

The openings of these channels, known as *Foramina Thebesii* are, however, very easily seen, particularly in the

auricles. That they communicate with the general venous system can readily be proved by injections. These experiments have been successfully carried out by Thebesius, Langer, and the author. Recently, the existence of *venae Thebesii* in the ventricles has been proved in a similar manner. This, however, has been denied by Nussbaum.

These vessels can be divided into two types: (a) With very small openings, 1–2 mm. in width, which drain the capillaries in the auricles; (b) with larger openings, in which several secondary openings can be seen, and which link up with large venous channels in the musculature and on the surface of the heart.

The *venae minimae Thebesii* are very numerous in the right auricles, being seen in greatest numbers in the interauricular septum, especially in the region of the *limbus Vieussenii* and *valvula Thebesii*. In the left auricle they are not so numerous, but generally larger. Thus, Langer has shown that, on the interauricular septum adjacent to the aortic valves, certain larger venous openings can be found which drain as well in part the superior portion of the interventricular septum.

It is possible that into the category of veins fall also the long grooves described by Lannelongue. If one of these is injected, the injection mass is forced out of the adjoining groove. Tandler does not feel that he can confirm with certainty Lannelongue's statement that these grooves drain small venous channels.

In the ventricles, *foramina Thebesii* are most frequent at the bases of the papillary muscles, the region of the *conus* on the right side, and, according to Langer, the apical musculature. They do not here communicate directly with the larger veins except possibly through capillary anastomoses,

and appear to be concerned more with draining the subendocardial spaces and the immediately adjacent musculature.

VARIATIONS IN THE VEINS OF THE HEART

As has already been stated, the venous structure of the heart is very liable to variations. Some of the more outstanding and interesting variations can easily be explained on an embryological basis.

Very early in embryonic life, the anterior and posterior cardinal veins link up to form the ductus Cuvieri, and these in turn open separately into the heart. Normally, only the right opening persists and this is made possible by the development of a great anastomosis between the anterior cardinal veins, which enables the left vein to conduct all its blood into the right auricle. The lower portion of the right anterior cardinal vein and the right ductus Cuvieri become the vena cava superior of the right side. The corresponding structure on the left side, as has been shown by Marshall, regresses so that only vestiges remain of the greater part of the left superior vena cava—namely, the vena obliqua atrii sinistri and the plica venae cavae; only the proximal portion of the left ductus Cuvieri is preserved as the sinus coronarius.

When the opening of the coronary sinus into the right auricle is obliterated, blood is carried through a persistent left superior vena cava into the vena innominata. This obliteration, as Siding has shown, must occur necessarily in the second month of embryonic life and at a time after the formation of the vena innominata.

Tandler cites 2 cases, that of Le Cat (1738) and that of A. Siding (1896), where this condition occurred. Here the sinus coronarius commences as a trunk which runs obliquely

over the posterior wall of the left auricle in front of the left pulmonary veins, through the ligamentum venae cavae to reach and empty into the vena innominata. The opening of the sinus coronarius into the right auricle is obliterated. Extending out from the right auricles, through an opening flanked by a low valvula Thebesii, is a blind sac about 10 mm. in length and representing the proximal portion of the sinus coronarius. The patent portion of the sinus coronarius, which continues up towards the pulmonary veins, receives the entrance of the vena coronaria sinistra and the vena interventricularis posterior through openings guarded by valves.

The above described condition is infrequent but deserves special mention on account of its interesting genesis.

A frequent variation and, according to Piquand who found it in 20 per cent of the cases, representing the primitive form, is the entrance of both vena coronaria sinistra and vena interventricularis posterior by a common short channel, the *truncus communis*.

In a number of hearts, the vena marginis acuti empties independently into the right auricle and is then called the *Vena Galeni*.

The vena coronaria dextra frequently varies in its site in the auriculoventricular groove, being often found lying above this in the auricular musculature.

Not infrequently a stout vena marginis obtusi ascends the surface of the ventricle and, curving parallel with the vena coronaria sinistra, opens independently into the sinus coronarius.

The venae minimae Thebesii are liable to much variation in their size, form and situation.

So far as structure is concerned, it has already been stated

that the sinus coronarius is usually surrounded by transverse muscular fibers from its origin to its conclusion at the valvula Vieussenii. The musculature may fall short of the valves but never, according to Tandler, passes beyond them.

CHAPTER VIII

AGE PERIOD CHANGES IN THE BLOOD SUPPLY TO THE HEART AND THEIR PATHOGENETIC RELATIONS

IN introducing the discussion on the age period changes which the blood supply to the heart undergoes, it may be permissible to quote, slightly modified, from the author's article on the postnatal evolution of the spleen, in order to make clear the general importance of the subject:

Various integral parts of the human organism die long before the whole organism is born. Birth and growth of new tissues continue long after the whole organism is born. Life, in fact, consists of the simultaneous breaking down and building up of protoplasm, cells, tissues and organs.

Embryonic development presents some of these phenomena in striking, almost dramatic, form. Moreover, it appears that these processes display, at least in some instances, a coordinated relationship.

Oertel was the first to show that the development and growth of the sex gland in the embryo is definitely correlated with the degeneration of the mesonephros, and thus gave an actual anatomic foundation to the idea that the relationship of cells, tissues and organs is by no means always altruistic, but, as held many years ago by Boll, Roux, Hansemann and others, often antagonistic.

Life and evolution of the individual, like life and evolution of any community, depend not only upon helpful but also upon opposing forces. These phenomena of embryonic life possess an additional interest to the pathologist because almost all pathological processes, degenerations, inflammations and even tumor growth, are, as Minot pointed out years ago, in principle and prototype to be found in the normal embryo.

In postnatal life, the appreciation of the unstable and constantly changing character of cells, tissues and organs has in recent years been somewhat more fully recorded especially since their pathogenetic importance has become clear. Thus,

since Reid's observations on the measurements of the heart and tables on the weight of the most important organs of the body at the different age periods appeared in 1843, several other important contributions on different organs have helped to show that these changes represent a general principle of life.

In 1883 Müller presented his observations on the weights and measurements on the heart and its structures, and though he did not particularly stress the age period idea, his carefully compiled statistics based on a very large number of hearts furnish, as will be shown later in the discussion, a great number of very interesting points.

It may be well, however, to outline here, first, the present knowledge regarding postnatal development in other organs of the body, thus showing the significance of age period changes and of the fluid state of tissue.

In 1904, Reitmann pointed out that the normal pancreas is, from infancy to old age, a very unstable organ. Degeneration, atrophy, cell loss and acinar collapse are, to a certain extent, normal performances, being almost simultaneously compensated for by formation of new cells, new acini and, in the growing, youthful pancreas, even by the formation of new lobules.

Coplin found equally great instability in the thymus, Theilhaber in the endometrium, and Milne in the ductless glands, chiefly the thyroid and suprarenal gland.

In 1909, Herxheimer called attention to the frequent occurrence of hyaline glomeruli in the kidneys of infants and young children. These, Oertel regards as remnants of reduced and regressing renal substance similar to those found in otherwise healthy adult kidneys without arterial disease.

Oertel, and Oertel and Anderson, have more recently been

able to corroborate and to extend Reitmann's observations on the pancreas and to show further that certain localized restricted abnormalities or faulty reconstructions may even normally be found, making it at times difficult to define on which side of the borderline between physiological and pathological they may be classified. Thus also the condition which Oertel has called "Essential Atrophy of the Pancreas" appears, as he states, to represent a pathological exaggeration of, and loss of balance in, normal, physiological processes of regression and progression which are constantly going on in the pancreas.

In his studies on degeneration, senescence and new growth, Oertel has further drawn attention to the close interrelation between these normally occurring processes and distinct pathological conditions. Somewhat similar observations have been made by René Sand in a study of pathological senescence.

In 1919, the author was able to add the spleen to the already growing list of organs showing a distinct cycle of progressive and retrogressive processes during the age periods, and pointed out that age has a most important bearing both as regards the anatomical structure of the spleen in health, as well as to the reaction which it presents in disease.

In 1920, Waugh came to similar conclusions on the bone-marrow and this was soon followed by Weed, whose studies convinced him that very definite qualitative structural modifications occur in the pia arachnoid in advanced age periods, notably in the appearance of newly formed endothelial cell clusters, etc.

Finally in 1921 Oertel summed up the existing facts on postnatal evolutions and pathological organ reconstruction in its relation to function and disease and came to the following conclusions:

(1) Organs which normally exhibit a developmental cycle of changes in cell elements and tissue organization, undergo corresponding functional modifications. Pathological, anatomical and functional changes must, therefore, be interpreted in conformity and comparison with an age period.

(2) Disposition to disease is influenced by structural alterations in tissue soil in the various age periods, especially in affording or not affording anchoring ground for bacteria. Moreover, it is likely that the fluid condition of organs influences the disposition to infections during waves of regression and progression as we already know it in unbalanced, juvenile and growing tissues.

(3) Anatomical and functional expressions of a disease vary in one and the same organ according to its construction and composition during an age period, and it would seem that these factors also exert an influence on variations in disposition and immunity. (The author has shown this to be true in endocarditis, and Lexer in osteomyelitis.)

(4) Diseases may arise from the fluid and changing state and age progress of organs by loss of balance in physiological regression and progression.

(5) Depending upon the pathological predominance of one or the other group of changes, essential atrophies, hypertrophies, degenerations and progressive, destructive cell proliferation, all of which are normal developmental processes, may be duplicated and exaggerated in post-natal existence.

Thus, Oertel has elevated the importance of postnatal age period changes to a general biological principle upon which depends the evolution of the individual from birth to senility and the development of disease.

One is now in a better position to appreciate similar changes in the heart, for the vascular architecture of the heart shows strikingly the remarkable effect of age periods in producing a cycle of events which register unmistakably their effects on function, both physiologically and pathologically. Moreover, these changes occur not only in the absolute sense, but also, as will be seen, in the relative constitution and construction of both sides of the heart.

Bizot has shown long ago that in the embryo both ventricles are about the same thickness; at birth the left is slightly thicker, and, after this, outstrips the right in growth so that it gradually becomes relatively more and more preponderant.

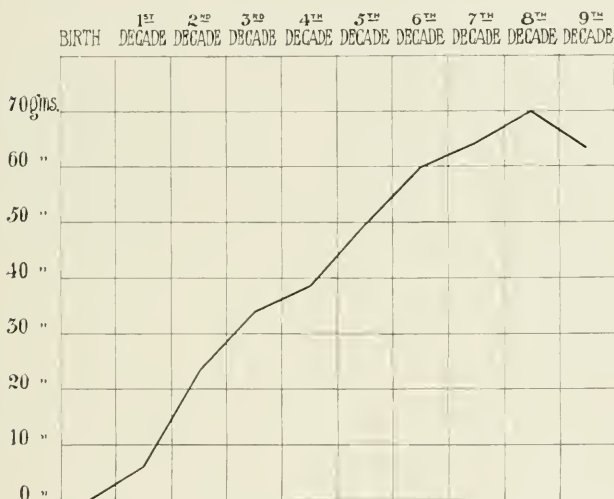


FIG. 16. — Graph showing the increase of subpericardial fatty tissue as age advances.

The development of fat under the pericardium he found to begin always at the base of the heart and to follow the right coronary artery to the apex, later the left coronary; finally the rest of the organs become the seat of fat deposit. The amount of subcutaneous fat bears no relation to the amount of subpericardial fat.

Valentin, Engel and Beneke studied the relationship between the right and left ventricles. Judging by their figures

there is an undoubted relatively greater increase on the left side, which reverses in the later decades of life.

Müller (1877-1881) made a study of 1,481 hearts. From observations on their gross weight he concluded that the turning point in growth of the heart is in the seventh decade in men and the eighth decade in women.

Though he held that there is a relative proportion between the amount of subpericardial and subcutaneous fat, his figures show a distinct and gradual increase in the former from birth to death. Figure 16 shows this process graphically in a chart which the author has plotted out from Müller's statistics.

A study of his figures on the comparison of the weight of the right and left auricle, in regard to age periods, shows that, on the whole, there is very little difference between the two.

His figures expressed in units of proportion (Table VIII) are as follows:

TABLE VIII

| Age | Male | | Female | |
|------------------|---------------|--------------|---------------|--------------|
| | Right auricle | Left auricle | Right auricle | Left auricle |
| Embryos..... | 2.2 | 1.5 | 1.9 | 1.4 |
| 1 Month..... | 1.7 | 1.5 | 1.7 | 1.4 |
| 2-12 Months..... | 1.6 | 1.7 | 1.6 | 1.6 |
| 2-15 Years..... | 1.7 | 1.6 | 1.8 | 1.7 |
| 16-20 Years..... | 1.7 | 1.6 | 1.8 | 1.6 |
| 21-80 Years..... | 1.8 | 1.7 | 1.8 | 1.7 |

From these he draws the following conclusions:

(a) The division of the auricular musculature of both auricles is different before and after birth. During the whole embryonic life, the muscle mass preponderates in the right auricle. During the first month after birth, the right auricle loses in weight. At the beginning of the second month, both are similar in weight. This equality more or less persists during the first year.

(b) From the second year of life until the period of maturation of sex, the left auricle preponderates, after which the right again preponderates.

Much more interesting and important in this connection are his figures comparing the mass of the right and left ventricles according to age periods (Table IX).

TABLE IX

| Age | No. of cases | Male | | | No. of cases | Female | | |
|--------------------|--------------------|-------|--------|----------|--------------------|--------|-------|----------|
| | | R. | L. | R. L. | | R. | L. | R. L. |
| Embryos 1-500 gms. | 10 | 0.42 | 0.36 | 0.845 | 6 | 0.37 | 0.39 | 0.731 |
| 501-1000 gms. | 9 | 1.13 | 1.17 | 0.769 | 7 | 1.29 | 1.40 | 0.710 |
| 1001-1500 gms. | 4 | 2.32 | 2.04 | 0.842 | 10 | 2.45 | 2.28 | 0.811 |
| 1501-2000 gms. | 11 | 3.41 | 3.06 | 0.864 | 11 | 3.06 | 2.59 | 0.902 |
| 2001-2500 gms. | 6 | 4.31 | 4.04 | 0.85 | 6 | 3.98 | 4.18 | 0.786 |
| 2501-3000 gms. | 6 | 6.02 | 4.84 | 0.925 | 7 | 5.46 | 4.92 | 0.849 |
| over 3000 gms. | 15 | 7.72 | 5.44 | 1.007 | 7 | 7.14 | 4.69 | 1.065 |
| 1 week post-natal | 16 | 4.85 | 4.45 | 0.839 | 17 | 3.82 | 3.47 | 0.827 |
| 2 weeks. | 13 | 4.11 | 4.79 | 0.698 | 15 | 4.10 | 4.53 | 0.733 |
| 3 weeks. | 10 | 4.10 | 4.93 | 0.680 | 5 | 4.04 | 5.04 | 0.678 |
| 4 weeks. | 5 | 4.11 | 5.83 | 0.635 | 10 | 3.44 | 4.71 | 0.638 |
| 2 months. | 14 | 3.09 | 4.54 | 0.594 | 14 | 3.43 | 5.42 | 0.571 |
| 3 months. | 14 | 3.94 | 6.44 | 0.561 | 16 | 3.88 | 6.41 | 0.545 |
| 4-6 months. | 24 | 4.68 | 7.99 | 0.532 | 20 | 4.33 | 7.91 | 0.522 |
| 7-12 months. | 34 | 5.72 | 10.68 | 0.502 | 31 | 5.77 | 10.43 | 0.515 |
| 2 years. | 17 | 9.00 | 14.11 | 0.561 | 24 | 7.82 | 13.52 | 0.525 |
| 3 years. | 13 | 10.63 | 23.77 | 0.469 | 16 | 9.04 | 18.26 | 0.473 |
| 4-5 years. | 17 | 11.07 | 22.23 | 0.473 | 19 | 11.71 | 21.94 | 0.499 |
| 6-10 years. | 16 | 17.68 | 33.98 | 0.487 | 21 | 14.31 | 21.92 | 0.471 |
| 11-15 years. | 8 | 24.20 | 44.40 | 0.500 | 9 | 20.10 | 40.90 | 0.467 |
| 16-20 years. | 23 | 46.00 | 76.90 | 0.542 | 13 | 30.10 | 73.80 | 0.508 |
| 21-30 years. | 69 | 54.10 | 90.50 | 0.519 | 46 | 37.90 | 72.90 | 0.499 |
| 31-40 years. | 67 | 50.80 | 88.90 | 0.529 | 57 | 37.70 | 68.90 | 0.509 |
| 41-50 years. | 82 | 51.70 | 94.90 | 0.506 | 69 | 45.20 | 75.20 | 0.552 |
| 51-60 years. | 84 | 54.90 | 101.60 | 0.508 | 58 | 43.30 | 73.90 | 0.529 |
| 61-70 years. | 87 | 55.10 | 103.70 | 0.516 | 83 | 46.60 | 81.20 | 0.545 |
| 71-80 years. | 62 | 52.40 | 94.40 | 0.526 | 61 | 43.90 | 82.70 | 0.515 |
| 81-90 years. | 11 | 41.20 | 97.20 | 0.442 | 12 | 36.20 | 66.10 | 0.488 |

Represented in chart form, the gradual and consistent, increasing, postnatal preponderance of the left over the right ventricle as age advances, is strikingly seen (Fig. 17).

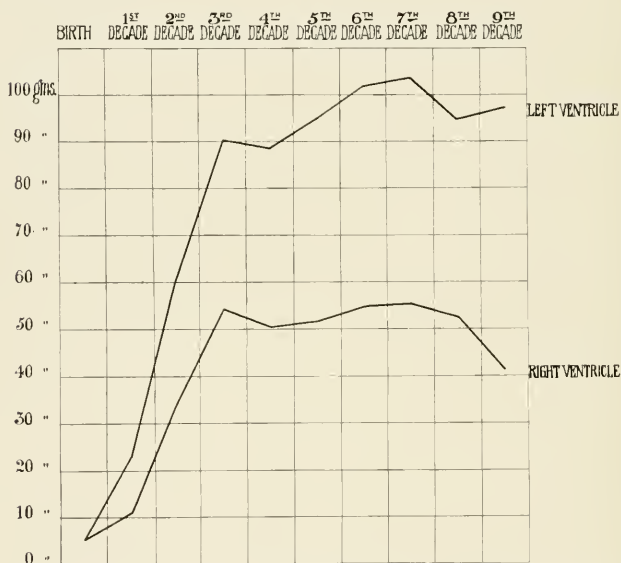


FIG. 17.—Graph showing the absolute increasing weight of the right and left ventricles as age advances, and also the relative increasing preponderance of left over right side.

Councilman has recently come to the conclusion that heart hypertrophy is very frequent in old people and regards it as pathological.

Lewis has been able to confirm Einthoven's observations that before birth and for several months after, the heart shows electrocardiographically a right-sided preponderance which

soon, however, becomes equal and from then on becomes preponderant in the left. Unfortunately, accurate comparative age period observations on this point are wanting for the post-natal development of the heart, but Lewis and others have observed electrocardiographic curves in adults which speak for left-sided hypertrophy in apparently normal cases. In old individuals, relative preponderance of the left ventricle is a common observation.

In consideration of all the foregoing facts, it is not surprising that the cardiac circulatory architecture shows a corresponding series of changes which, in their qualitative and quantitative nature, are of prime physiological and pathological importance.

If one examines Figure 18, which represents a roentgenogram of the circulation in an average heart at birth, it is seen that both sides of the heart are equally divided and supplied with blood, so that, were it not for the characteristic ramus circumflexus dexter, one would be at a loss to discern the left from the right side. Examined stereoscopically, this heart fails to show macroscopic septal anastomoses. The branches show a uniformity of lumen, and are, with the exception of their extremities, on the whole without tortuosity.

Figure 19 is a photograph of injected and cleared specimens of hearts at birth. It will be seen that at this period of life no arteriae telae adiposae are visible in the subpericardial fat, so that the auriculoventricular sulci carry no fat-vessels, nor do these accompany the main coronary branches.

Figure 20 represents an average heart of the first decade. In this roentgenogram the main branches pursue a straight and even course. There is a beginning clearing of the right side. Septal anastomoses cannot yet be made out. This heart did not show,

upon examination of the cleared specimen, any arteriae telae adiposae.

In the second decade (Fig. 21), the distribution of blood is beginning to be a little more marked on the left side. The vessels have hardly commenced to show their tortuosity. The stereoscope, however, already reveals very delicate septal anastomoses. At the furrows, the cleared specimen shows a few stray fat-vessels, best seen as rami telae adiposae parallel to the main branches.

The third decade of life (Fig. 22) shows a definite, though not yet marked, left-sided vascular preponderance. Septal anastomoses are now much more clearly made out. The tortuosity of the vessels is quite discernible and, in the cleared specimen, rami telae adiposae are well seen.

The fourth decade of life presents these changes in definite progress (Fig. 23). The septal anastomoses are quite clearly developed. The left side of the heart is definitely in the ascendant. Tortuosity of vessels is clearly seen and becoming marked.

Figure 24 is a photograph of an average injected and cleared heart from this decade. It shows the already well-developed fat-vascular system. It will be seen that the anterior surface of the heart displays in the auriculoventricular groove a striking network of delicate arteriae telae adiposae. At the edges of the heart these are seen projecting from the surface into the subpericardial fat. The parallel fat-vessels can quite easily be made out as they accompany the main branches, particularly the ramus descendens anterior.

Figure 25 shows the posterior surface of the same heart. Here, there is displayed even better, the well-developed network of arteriae telae adiposae. It fills the whole auriculoventricular groove and appears as a greyish maze of vascular

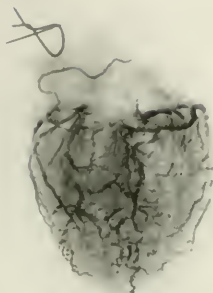


FIG. 18.—Roentgenogram of the blood supply in the average heart at birth.

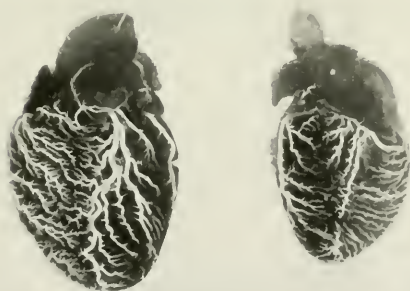


FIG. 19.—Photograph of injected and cleared specimen, showing the superficial distribution of the coronary arteries at birth.



FIG. 20.—Roentgenogram of the blood supply in the average heart of the first decade.



FIG. 21.—Roentgenogram of the blood supply in the average heart of the second decade.



FIG. 22.—Roentgenogram of the blood supply in the average heart of the third decade.



FIG. 23.—Roentgenogram of the blood supply in the average heart of the fourth decade.



FIG. 24.—Photograph of the anterior surface of an injected and cleared heart of the fourth decade, showing the distribution of the *arteriae telae adiposae*.



FIG. 25.— Photograph of the posterior surface of the same heart as in Fig. 24, showing the distribution of the arteriae telae adiposae.

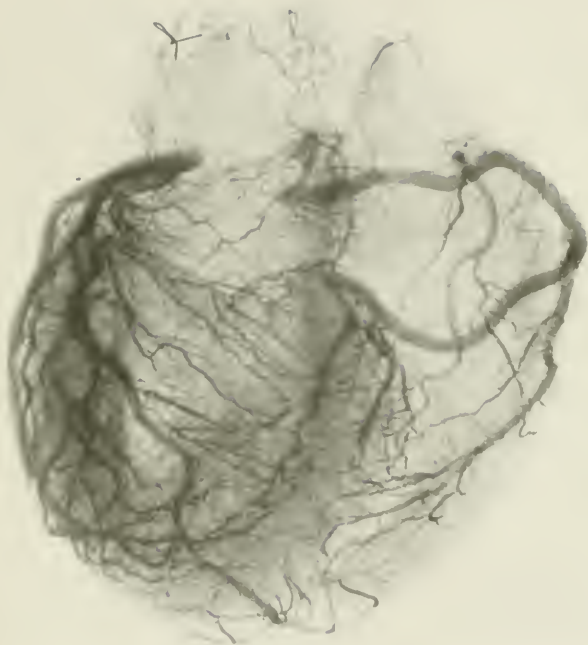


FIG. 26.—Roentgenogram of the blood supply in the average heart of the fifth decade.



FIG. 27.—Roentgenogram of the blood supply in the average heart of the sixth decade.

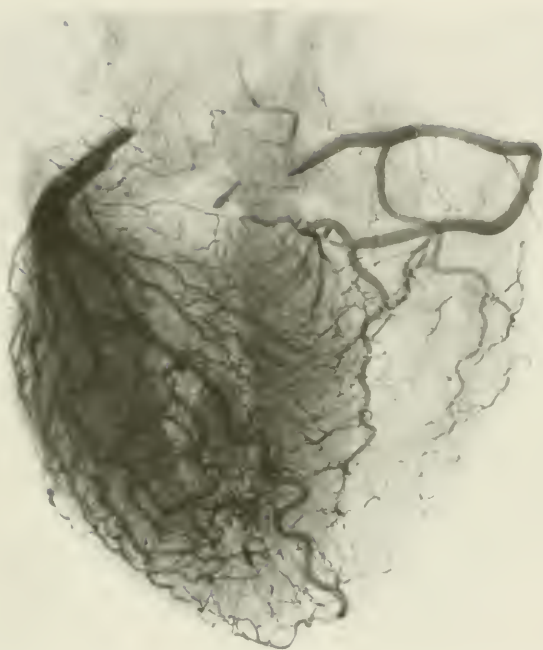


FIG. 28. Roentgenogram of the blood supply in the average heart of the seventh decade.



FIG. 29.—Roentgenogram of the blood supply in the average heart of the eighth decade.

channels. Here too, the accompanying fat-vessels are clearly seen.

In the fifth decade (Fig. 26) the preponderance of left side over right is striking, as is also the tortuosity of the vessels. At this period of life the main branches are occasionally seen (as they are in this instance) projecting beyond the mass of heart musculature into the fat. This is due to a beginning regression and atrophy of heart muscle, leaving the vessels relatively too long. The septal anastomoses are distinct and abundant, being arranged somewhat in the fashion of a row of harp strings. In the cleared specimen, fat-vessels are quite well developed and numerous.

The sixth decade of life (Fig. 27) shows an ever increasing left-sided vascular preponderance and tortuosity of the vessels. The septum shows a system of very patent and free arterial anastomoses. The increase of the *rami telæ adiposæ* is in keeping with the other changes.

Figure 28 shows very beautifully the stage of development of these four features in the seventh decade of life; the increasing relative anemia of the right side, the marked tortuosity of the vessels, the rich and abundant septal anastomoses and, in the cleared specimen, the well-developed fat-vessel network.

In the eighth decade of life another complicating feature appears. In a large percentage of cases arteriosclerotic changes are seen. This is well shown in Figure 29. Here the *ramus circumflexus dexter* presents in its entire extent an unevenness of bore, and at its termination just before the ultimate branching, a distinct aneurysmal bulging. Moreover, the left-sided vascular preponderance, the tortuosity, the *rami telæ adiposæ* and, in the stereoscopic plates (for this plate was taken at right angles with the direction of the septum, hence shows

anastomoses only upon stereoscopic examination), the septal anastomoses are seen to be distinctly on the increase.

Figure 30 is a composite, representing the contrast between the circulation of the heart at birth and in the seventh decade of life. Here one sees exceedingly well illustrated the difference in the relative amounts of blood supplying both sides of the heart, in the tortuosity of the vessels and the patency of the septal anastomoses. A comparison of Plates 19, 25 and 34 shows the progressive and consistent increase of the rami telae adiposae from birth to the fourth, and from this, to the eighth decade of life.

Perhaps there is no better way of concretely establishing the importance of these changes in the postnatal evolution of the cardiac vasculature than by observing their effects upon function.

Figure 31 is a roentgenogram of a heart from a female, aged seventy-three, who died of cancer of the gall-bladder. During life she had no symptoms referable to the heart or coronary vessels. In the hospital no signs were found during life indicating a lesion of the heart. Upon injection of the heart at autopsy, however, it was seen that the ramus circumflexus dexter presented to a great extent of its course an arteriosclerotic obliteration which was, in several places, almost complete. The myocardium was absolutely intact, no suggestion of an infarct being present. The case is, in short, very similar to that described by Galli. A more careful observation of the plate shows that a very ample and abundant anastomosis of large, patent rami interventriculares supplied, to a great extent, the right side, and this was further compensated by an extensive labyrinthine felt-work of rami telae adiposae which massively covered the right ventricular surface. (Fig. 32

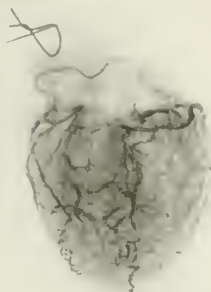


FIG. 30.—Roentgenogram of the blood supply in the average heart at birth and in the seventh decade, illustrating the marked evolutionary changes which the advancing age periods have produced.



FIG. 31.—Roentgenogram of an infected heart, showing the results of a well-marked and almost complete arteriosclerotic obliteration of the right coronary artery.



FIG. 32.—Photograph of injected and cleared specimen, showing the development of the rami telae adiposae on the right side of the heart, illustrated in Fig. 31.

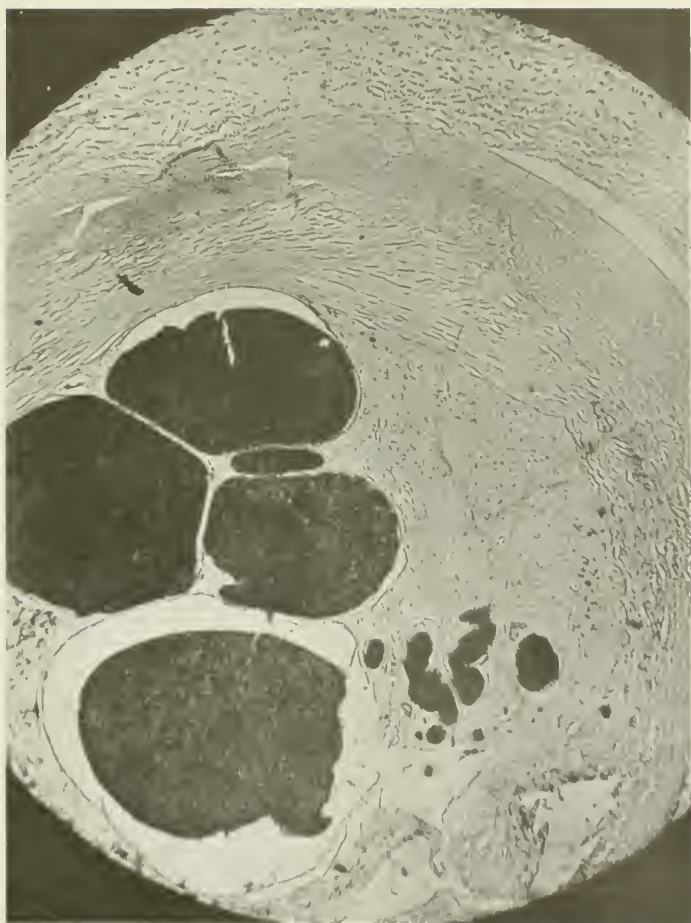


FIG. 33.—Microphotograph of a section through the arteriosclerotic vessel from the heart, illustrated in Fig. 31.

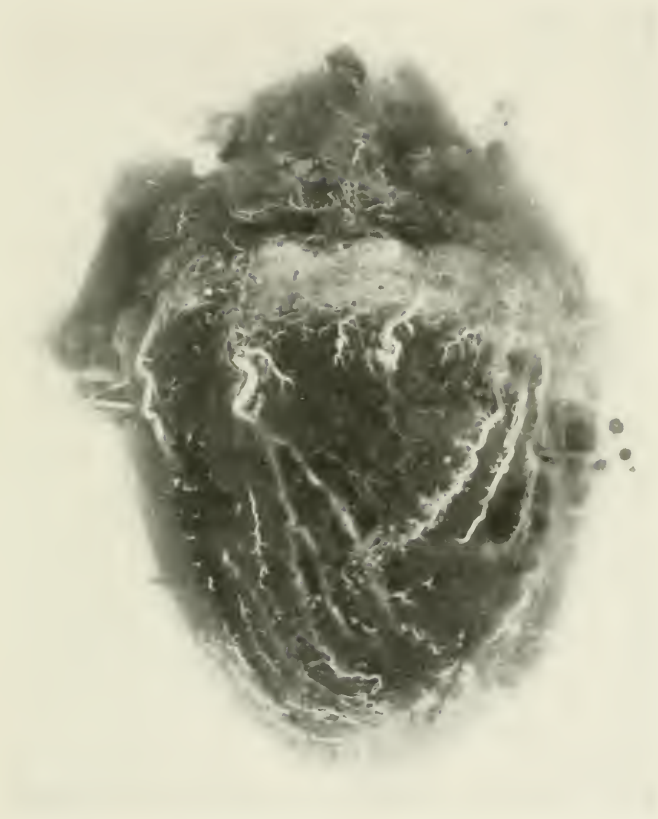


FIG. 34.—Photograph of injected and cleared specimen, showing the rami telae adiposae on the left side of the heart, illustrated in Fig. 31.

shows a photograph of this injected and cleared specimen.) These fat-vessels were traced into the musculature and were found to anastomose with persistent right branches and with the compensating left ones.

It will be observed in the roentgenogram that the ramus ostii cavae superioris pursues an undisturbed course to the sino-auricular node because the arteriosclerotic process commences just beyond the origin of the vessel from the right coronary artery.

Figure 33 shows a microscopic section through one portion of the partially obliterated artery. It is seen that extensive obliterating endarterial changes have left a few canalized areas through which the barium (black in the photograph) has forced its way. In other places the obliteration was even more marked.

The conclusions which can be drawn from this case are, that in the seventh decade of life the vascular architecture of the heart is well prepared to receive the brunt of the obliteration even of a main coronary artery, not only on account of the existence of abundant and free anastomoses, but also, as is seen in this case, by the non-negligible factor of the development at this age period of a dense felt-work of arteriae telae adiposae which can compensate and supply considerable blood to the subjacent muscle. In fact, that the rami telae adiposae are able to increase in caliber and quantity so as to furnish additional blood, is seen by comparing Figure 32 with Figure 34, (which represents the posterior surface of the same heart and shows a fat-vessel structure commensurate with this age period). This comparison shows that the increase of fat-vessels on the right side is out of all proportion to the normal development, but it must be remembered that this great increase is only

possible on a basis of an already well-developed structure. Furthermore, in this particular case, the gradual obliteration allowed for an ample development of these compensating structures, so that the myocardium was left absolutely intact.

There are all gradations of possibilities which lead up to this interesting case. It has already been shown in Chapter VI that the heart possesses in the anastomoses a structure which can and does give the heart considerable vascular reserve. This, moreover, as appears from our present discussion, becomes gradually more and more valuable for such a purpose as age advances, and since the heart presents at the same time a parallel increase of *rami telae adiposae*, a very potent ally, it acquires a compensating structure whose functional possibilities increase in direct proportion with age, that is, with that time of life when pathological processes would make an increasing, more frequent and greater demand upon it. There is thus a definite functional significance in these progressive evolutionary changes.

That the *rami telae adiposae* are an expression of a general body nutritional reserve structure would seem to be indicated by the fact that they have been found by the author in other organs which show a senile increase of fat. Thus, for example, in the senile as well as in the contracted kidney, the very fatty pelvic contents possess a rich supply of *arteriae telae adiposae*. In the contracted kidney, they can distinctly be seen penetrating the pyramidal parenchyma and supplying it with blood.

It appears then, that there is a definite reason for the progressive increase of subpericardial fat as shown by Figure 16, made from Müller's figures, and that this serves as a carrier

for a most important and apparently hitherto unrecognized functional and compensating unit.

It cannot be argued that the development of fat-vessels is entirely secondary to functional need, for it has already been shown that their increase is progressive and proportional to age, irrespective of the presence of a pathological lesion. It is true, however, that an obliteration of a coronary branch can bring about an added development and quantitative as well as qualitative changes in them.

The increasing tortuosity of the vessels, a general expression of increasing age, can be accounted for by the qualitative deterioration in the vascular wall and by a relative shrinkage and atrophy of the heart muscle in the later decades of life.

There remains, therefore, only to explain the gradually developing relative right-sided anemia. It must be borne in mind, first of all, that this is to a great extent relative; the increasing left-sided musculature and consequently vasculature overshadow that of the right side. Furthermore, the right ventricle perhaps does not so much present a regression of vessels as a falling behind in circulatory development, so that it becomes relatively more and more anemic.

Without doubt, these changes are of far-reaching functional significance. It is easy to comprehend why in embryonic life the right side, which is the more actively functioning one, should attain a greater development of its vascular tree. This persists for some time after birth, but with the assumption of greater activity by the left ventricle, the latter becomes more and more richly supplied with blood; whereas the right ventricle, through less activity and lesser importance, begins to lag in its vascular development, a process which apparently progresses consistently throughout life.

As old age approaches, the individual is ushered into an era of many dangers through right-sided heart decline. Thus, the possibility from death by right-sided heart paralysis in infectious diseases increases with age, and death from pneumonia in old age, so frequent that it is practically "physiological," comes perhaps somewhat nearer to our comprehension when we consider that this lagging right-sided circulation, reacting upon the right ventricle, produces a physiological right-sided decline or increasing heart failure. The lung tissue, which depends largely upon the blood from the pulmonary artery for its nutrition, receives an increasingly sluggish supply and becomes, therefore, a more and more suitable soil for a terminal infection. (Oertel states that in a long autopsy experience of almshouse cases, many of which concerned, of course, senile decrepits, it was not infrequent to discover unsuspected pneumonias in sudden deaths; for example, in individuals who had retired at night in apparent relative health and were found dead next morning. This has also a certain medicolegal interest.)

Perhaps it is, therefore, permissible to paraphrase in this connection the old adage about a man being as old as his arteries, so as to read, "A man is as old as his *Right Coronary Artery*."

BIBLIOGRAPHY

- ABBOTT, MAUDE E. Monograph on congenital cardiac disease. Osler and McCrac's Modern Medicine, Philadelphia, 1915, iv.
- ACKER, R. B. Delayed death in coronary thrombosis. *J. A. M. M. Assn.*, 1919, lxxiii.
- ALBRECHT. Der Herzmuskel und seine Bedeutung für Physiologie, Pathologie und Klinik des Herzens, Berlin, 1903.
- AMENOMIYA. Über die Beziehungen zwischen Koronararterien und Papillarmuskeln im Herzen. *Virchow's Arch. f. path. Anat.*, Berl., 1910, cxcix.
- ASCHOFF. (1) Über den Glykogengehalt des Reizleitungssystems des Säugetierherzens. *Verbandl. d. deutsch. path. Gesellsch.*, Berl.; Jena, 1908, xii.
- (2) *Deutsche med. Wchnschr.*, Berl. and Leipz., 1908, No. 9.
- BANCHI. (1) Le variazioni delle arteriae coronariae cordis et la morphologia di questi vasti. *Sperimentale. Arch. di biol.* Firenze, 1903, lvii.
- (2) Morfologia delle arterie coronariae cordis. *Arch. ital. di anat. e di embriol.*, Firenze, 1904, iii.
- BARDELEBEN. Lehrbuch der systematischen Anatomie des Menschen, Berlin-Wien, 1906.
- BARKER and HIRSCHFELDER. Effects of cutting branch of the His bundle going to left ventricle. *Arch. Int. Med.*, 1909, iv.
- BAYNE-JONES. (1) Blood-vessels of the heart valves. *Am. J. Anat.*, 1917, xxi.
- (2) *Johns Hopkins Hosp. Rep.*, 1919, xviii.
- BENEKE, F. W. (1) Die anatomischen Grundlagen der Constitutionsanomalien des Menschen, Marburg, 8, 18-8.
- (2) Über das Volum des Herzens und die Umfänge der grossen Arterien des Menschen. *Schriften der Gesellschaft zur Beförderung der gesamten Naturwissenschaften zu Marburg*, 1881, xi, 4, Suppl. 2, Kassel.
- BERNAYS. *Morphol. Jahrb.*, Leipz., ii.
- BÉRAUD. Insertions supérieures du péricarde. *Gaz. méd. de Par.*, 1862.
- BETTELHEIM, K. Über die Störungen der Herzmechanik nach Compression der Arteria coronaria sinistra des Herzens. *Ztschr. f. klin. Med.*, Berl., 1892, xx.

- BEZOLD, A. VON. Von den Veränderungen des Herzschlages nach Verschlussung der Koronararterien. *Untersuchungen aus dem physiologischen Laboratorium zu Würzburg*, 1867, ii.
- BEZOLD, A. VON and BREYMAN, E. Von den Veränderungen des Herzschlages nach Verschlussung der Koronarvenen. *Untersuchungen aus dem physiologischen Laboratorium zu Würzburg*, 1867, ii.
- BIZOT, J. Recherches sur le coeur et le système artériel chez l'homme. *Mémoires de la Société méd. d'observation*, Paris, 1837, i, 8.
- BOCHDALEK. Zur Anatomie des menschlichen Herzens.
 (1) Über die sogenannte pars membranacea septi ventriculorum cordis.
 (2) Über die foramina Thebesii. *Arch. f. Anat. (Anat. Abt.)*, Leipz., 1868.
- BOURCERET. Les circulations locales: la main, Paris, 1885, xiii.
- BRÄUNIG, K. Über muskulöse Verbindungen zwischen Vorkammer und Kammer des Herzens. *Berl. klin. Wchnschr.*, xli.
- CADIAT. Étude sur l'anatomie et la physiologie du coeur. *Bull. Acad. de méd.*, Par., 1879, viii.
- CALDANI, L. M. A. and CALDANI, F. Icones anatomicae, etc., iii, 1810, Venetia, 1801-14.
- CHIARI. (1) Über Netzbildungen im rechten Vorhofe des Herzens. *Beitr. z. path. Anat. u. z. allg. Path.*, Jena, 1897, xxii.
 (2) Thrombotische Verstopfung der rechten und embolische Verstopfung des Hauptstammes der linken Koronararterie des Herzens bei einem 32-jährigen Manne. *Prag. med. Wchnschr.*, 1897.
- CHIRAC, P. De motu cordis adversaria analytica, Monspelii, 1698.
- COËN. Über die Blutgefäße der Herzklappen. *Arch. f. mikr. Anat.*, 1886, xxvii.
- COHN. On the auriculonodal junction. *Heart*, Lond., 1900-10, i.
- COHNHEIM and VON SCHULTHESS-RECHBERG, A. Über die Folgen der Kranzarterienverschlussung auf das Herz. *Virchow's Arch.*, Berl., 1881, lxxxv.
- COTTON. *Heart*, Lond., 1915-17, vi.
- COUNCILMAN. The conditions presented in the heart and kidneys of old people. Contributions to Medical and Biological Research, Dedicated to Sir William Osler, in Honor of his 70th Birthday, New York, 1919, ii.
- CRUVEILHIER. Anatomie descriptive, iii, 1834, Traité d'Anatomie descriptive, Paris, 1844.
- CURRAN. (1) A constant bursa in relation with the bundle of His; with studies of the auricular connection of the bundle. *Anat. Anz.*, Jena, 1900, xxxv.
 (2) *Anat. Record.*, Phila., 1909, iii.

- DARIER. Les vaisseaux des valvules du coeur chez l'homme à l'état normal et à l'état pathologique. *Arch. de Physiol.*, Par., 1888, Nos. 5 and 6.
- DOGIEL. Die Bedingungen der automatisch-rhythmischen Herzcontractionen. *Arch. f. d. ges. Physiol.*, Leipz., cxxxv.
- DRAGNEFF. Recherches anatomiques sur des artères coronaires du coeur chez l'homme, Thèse, Nancy, 1897.
- ENGEL, J. (1) Über einige pathologisch-anatomische Verhältnisse des Herzens. *Wien. med. Wchnschr.*, 1863, xiii, Nos. 44, 45, 46, 51.
 (2) Über Organgewichte in Krankheiten. *Ztschr. d. k. k. Gesellsch. d. Ärzte in Wien*, 1865, xxi, No. 2.
 (3) Gewichtsbestimmungen an Leichen. *Wien. med. Wchnschr.*, No. 63, 1869.
- ENGELMANN. Fall von Mangel einer Koronararterie. *Anat. Anz.*, Jena 1898.
- ERICHSEN. On the influence of the coronary circulation on the action of the heart. *Med. Gaz.*, Lond., 1842, ii.
- ERLANGER. (1) The physiology of heart-block in mammals. *J. Exp. M.*, 1906, viii.
 (2) Can functional union be reestablished between mammalian auricles and ventricles after destruction of a segment of the auriculoventricular bundle? *Am. J. Physiol.*, 1909, xxiv.
- ERLANGER and BLACKMAN. A study of relative rhythmicity and conductivity in various regions of the auricles of the mammalian heart. *Am. J. Physiol.*, 1907, xiv, No. 1.
- FAHR. (1) Zur Frage der atrioventrikulären Muskelverbindung im Herzen. *Verhandl. d. deutsch. path. Gesellsch.*, 1908, xii.
 (2) *Virch. Arch.*, Berl., 1907, clxxxviii.
- FALLOPIUS. *Medici mutinensis observationes*, Venetüs, 1562.
- FENOGLIO and DROGUÉLL. Observations sur l'occlusion des coronaires cardiaques. *Arch. ital. de biol.*, Roma, ix.
- FINLEY. Cardiac disease with extensive venous and cardiac thrombosis. *Canad. M. Assn. J.*, 1910, ix.
- FLACK. Sinoauricular node. *J. Physiol.*, Lond., 1910-11, xli.
- FORSTER. Zur Frage der Formveränderungen der Herzmuskelkerne. *Deutsch. Arch. f. klin. Med.*, Leipz., 1906, lxxxi.
- FREY. *Handbuch der Histologie und Histochemie*, Leipzig, 1867, 2nd ed.
- FREYETT. *Arch. Roentg. Ray*, Lond., Dec., 1905, x, Plate 120.
- FUJINAMI. Über die Beziehungen der Myokarditis zu den Erkrankungen der Arterienwandungen. *Virchow's Arch.*, Berl., 1900, clix.
- GALLAVARDIN and DUFONT. Embolie de l'artère coronaire antérieure

- avec bradycardie à 22-28; contribution à l'étude de la mort rapide par obliteration coronaire. *Lyon Med.*, 1913, cxxi.
- GALLI, G. Über anastomotische Zirkulation des Herzens. *München. med. Wchnschr.*, L, No. 27.
- GASKELL. On the innervation of the heart. *J. Physiol.*, 1883, iv.
- GEGENBAUR. Notiz über das Vorkommen der Purkinjeschen Fäden. *Morphol. Jahrb.*, Leipz., 1877, iii.
- GERARD. Sur l'existence de canaux anastomotiques artérioveineux. *Arch. de physiol.*, Par.,vii, Ser. 5.
- GERLACH. Handbuch der allgemeinen und speciellen Geweblehre, Mainz, 1848.
- GLEW, F. H. *Arch. Roentg. Ray*, Lond., 1899, iv.
- GOUGH. A method of injecting the blood-vessels for roentgenological studies and simultaneously embalming. *Anat. Record*, March, 1920, xviii.
- GOUGUENHEIM and MENARD. Rupture spontanée du ventricule gauche; thrombose de la coronairée gauche; athérome généralisé. *Bull. et mém. Soc. méd. d. hôp. de Par.*, 1878-1879, xv, 38-40.
- GROBER. Massenverhältnisse des Herzens bei künstlicher Arterienstarre. *Verbandl. d. Kong. f. innere Med.*, Wien, 1907, xxiv.
- GROSS, LOUIS. (1) Studies on the circulation of the kidney. First communication. *J. Med. Research*, July, 1917, xxvi, No. 3.
- (2) On methods of organ reconstruction by injection of the arterial tree. *Internat. Assn. Med. Mus., Bull.*, Wash., May, 1918, No. 7, pp. 33.
- (3) Studies on the circulation of the kidney. Second communication. *J. Med. Research*, July, 1918, xxxviii, No. 3.
- (4) Studies on the gross and minute anatomy of the spleen in health and disease. *J. Med. Research*, January, 1919, xxxix, No. 3.
- (5) Preliminary report on the reconstruction of the circulation of the liver, placenta and lung in health and disease. *Canad. Med. Assn. J.*, July, 1919.
- GRUBER. (1) Über den Sinus Communis und die Valvulae der Venae cardiacae und über die Duplizität der Vena Cava Superior bei dem Menschen und den Säugetieren, St. Petersburg, 1864.
- (2) Über den Sinus Communis und die Valvulae der Venae cardiacae. *Mém. de l'Acad. des Sc. de St. Petersburg*, 1867.
- GUSSENBAUER. *Sitzungsb. d. k. k. Akad. d. Wissensch.*, Wien, lvii.
- GUTHRIE and PIKE. Activity of excised mammalian heart. *Am. J. Physiol.*, 1907, xviii.
- HAAS, G. Über die Gefäßversorgung des Reitzleitungssystems des Herzens. Inaug. Diss., Freiburg i. B., July, 1911.

- HALBERTSMA. *Ontleedkundige antekeningen. Nederl. Tijdschr. v. Geneesk.*, Amst., 1863, Tweede Zestel, 23.
- HALL. On the mutual relations between anatomy, physiology, pathology and therapeutics. *Gulstonian Lectures*, read at the British Association for the Advancement of Science, June 24, 1842.
- HALLER. *Elementa physiologiae corporis humani*, Lausannae, 1757.
- HAUCH. Die Arterien der gesunden und kranken Niere im Roentgengebilde. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, Hamb., 1903, xx.
- HEBERDEN. *Commentaries on the History and Cure of Disease*, London, 1816, 4th ed., Chap. lxx.
- HENLE. (1) *Handbuch der systematischen Anatomie des Menschen*, Braunschweig, 1866, 1st ed., iii.
 (2) *Handbuch der systematischen Anatomie des Menschen*, Braunschweig, 2d ed., 1873, iii.
 (3) *Handbuch der Gefässlehre des Menschen*, Braunschweig, 1876, iii.
- HERING. (1) Über die mikroskopischen Ausgangspunkte der Ursprungsreize des Säugethierherzens. *Arch. f. d. ges. Physiol.*, Bonn, 1906, cxvi.
 (2) On the beginning of the contraction of the papillary muscles and their relationship to the arterioventricular bundle. *Zentralbl. f. Physiol.*, Leipz. and Wien, 1908, xxi.
 (3) Papillary muscles and relationship to the arterioventricular bundle. *Arch. f. d. ges. Physiol.*, Bonn, 1909, cxxvi.
- HERRICK. (1) Clinical features of sudden obstruction of the coronary arteries. *J. Am. M. Assn.*, 1912, lix.
 (2) *Tr. Assn. Am. Physicians*, Phila., 1912, xxvii, 100-116.
 (3) *Med. Rec.*, 1918, xciv.
 (4) *Tr. Assn. Am. Physicians*, Phila., 1918, xxxiii.
 (5) Thrombosis of the coronary arteries. *J. Am. M. Assn.*, 1919, lxxii.
- HERXHEIMER. Hyaline Degeneration der Glomeruli der Niere. *Ziegler's Beitr. z. path. Anat. u. z. allg. Path.*, Jena, 1909, xlv.
- HEWSON, A. *Proceedings of the Pathological Society of Philadelphia*, 1915.
- HILDEBRAND. Über die Methode durch Einbringen von schattengebenden Flüssigkeiten Hohlorgane des Körpers im Röntgenogram sichtbar zu machen. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, Hamb., 1907, ix.
- HILDEBRAND, SCHULZ and WIEFING-PASCHA. Das Arteriensystem des Menschen im stereoskopischen Röntgenbild, Wiesbaden, 1911.
- HIRSCH and SPALTEHOLZ. Koronararterien und Herzmuskelarterien. *Deutsche med. Wchnsch.*, Berl. and Leipz., 1907, xx.
- HIS, JR. (1) Die Tätigkeit des Embryonalen Herzens und deren Bedeutung für die Lehre von der Herzbewegung beim Erwachsenen. *Arch. a. d. med. Klin. zu Leipzig*, 1893.

- (2) Herzmuskel und Herzganglien. *Wien. med. Bl.*, 1894.
- HOFFMAN. Beitrag zur Kenntniss der Purkinjeschen Fäden im Herzmuskel. *Ztschr. f. wissensch. Zool.*, Leipzig, 1902, lxxi.
- HOWELL. Textbook of Physiology, Philadelphia, 2nd ed., 1900.
- HUBER. Einfluss der Kranzarterienerkrankung auf das Herz. *Virchow's Arch.*, Berl., 1882, lxxxix.
- HUCHARD. Maladie du coeur et des vaisseaux, artériosclérose aortites, cardopathies artérielles, angines de poitrine, etc., Paris, 1889.
- HUNTER, JOHN. A treatise on the blood, inflammation and gunshot wounds, London, 1794.
- HYRTL. (1) *Wiener Sitz-Ber.*, xiv, 73, 1855.
 (2) Die Corrosionsanatomie und ihre Ergebnisse, Wien, 1873.
 (3) Anatomie des Menschen, Wien, 1878.
- JAMIN and MERKEL. Die Koronararterien des menschlichen Herzens in stereoskopischen Röntgenbildern, Jena, 1907.
- JELICK, COOPER and OPHÜLS. The Adams—Stokes Syndrome and the Bundle of His. *J. Am. M. Assn.*, 1906, xvi, 955.
- JOSEPH. Über die Ringe und Klappen des menschlichen Herzens. *Virchow's Arch.*, Berl., 1858, xiv.
- KEITH. (1) The auriculoventricular bundle of the human heart. *Lancet*, 1906, ii.
 (2) The form and nature of the muscular connection between the primary divisions of the vertebrate heart. *J. Anat. and Physiol.*, Lond., 1907, xli.
- KEITH and MACKENZIE. Recent researches on the anatomy of the heart. *Lancet*, 1910, i.
- KENT. Mammalian heart. *J. Physiol.* Lond., 1893, xiv.
- KOCH. (1) Über das Ultimatum moriens des menschlichen Herzens. *Ziegler's Beitr. z. path. Anat. u. z. allg. Path.*, Jena, 1907, xlii.
 (2) Über die Blutversorgung des Sinusknotens und etwaige Beziehungen des letzteren zum Atrioventrikularknoten. *München. med. Wchnschr.*, 1909.
 (3) Über die Struktur des oberen Cavatrichters. *Deutsche med. Wchnschr.*, Berl. and Leipzig, 1909, No. 10.
 (4) Weitere Mitteilung über den Sinusknoten des Herzens, *Verbandl. d. deutsch. path. Gesellsch.*, Leipzig, 1909, xiii.
- KÖLLIKER. Handbuch der Gewebelehre, Leipzig, 1902, ii.
- KÖLSTER. Experimentelle Beiträge zur Kenntniss der Myomalacia Cordis. *Skandin. Arch. f. Physiol.*, Leipzig, 1892, iv.
- KÖNIGER. Histologische Untersuchungen über Endokarditis. *Arbeiten aus dem Pathologische Institute zu Leipzig*, 1903–1908.
- KÖSTER. (1) Bericht über die Verhandlung der pathologischen Anatomie. Sektion auf d. 10 internat. med. Kongress zu Berlin, 1890. *Zentralbl. f. norm. u. path. Anat.*, Berl. and Wien, i.

- (2) Die embolische Endocarditis. *Virchow's Arch.*, Berl., lxxii.
- (3) Einige Fragen zur Anatomie und Physiologie des Herzens. *Verhandl. d. naturhist. Ver. d. preuss. Rheinlande u. Westphalens*, i.
- KRAUSE, C. F. Handbuch der menschlichen Anatomie, Hanover, 1842.
- KRAUSE, W. Die Blutgefäße der Herzklappen. *Internat. Monatschr. f. Anat. u. Physiol.*, vi.
- KRAUS and NICOLAI. Über das Elektrocardiogram unter normalen und pathologischen Verhältnissen. *Berl. klin. Wchnschr.*, 1907, xlv.
- KÜRSCHNER. (1) Über die venösen Klappen des Herzens und ihre Aktion. *Notiz. a.d. Geb. d. Nat.-u. Heilk. (Froriep's)*, Weimar, 1840, No. 316.
- (2) Herz und Herzfähigkeit. Wagner's Handwörterbuch, *Braunschweig*, 1884, v.
- LANGER. (1) Anastomosen der Koronararterien. *Sitzungsab. d. k. k. Akad. d. Wissensch.*, Wein, 1880.
- (2) Die Foramina Thebesii im Herzen des Menschen. *Sitzungsab. d. k. k. Akad. d. Wissensch.*, Wein, 3 Abt., 1880, lxxxii.
- (3) Über die Blutgefäße der Herzklappen. *Sitzungsab. d. k. k. Akad. d. Wissensch.*, Wein, 1880, lxxxii.
- (4) Über die Blutgefäße der Herzklappen bei Endocarditis Valvularis. *Virchow's Arch.*, Berl., 1887, cix.
- LANNELONGUE. (1) Circulation veineuse des parois auriculaires du coeur, Thèse, Paris, 1867.
- (2) Recherches sur la circulation des parois du coeur. *Arch. de physiol. norm. et path.*, Par., 1868, i.
- LE CAT. Histoire de l'Académie royale des sciences, Amsterdam, 1738.
- LEUCHTWEIS. *Deutsch. Arch. f. klin. Med.*, Leipz., lxxxvi.
- LEWIS, THOMAS. (1) The experimental production of paroxysmal tachycardia and effect of ligation of the coronary arteries. *Heart*, Lond., 1909-10, i, 43.
- (2) *Brit. M. J.*, 1912, i.
- (3) Clinical Electrocardiography, New York, 1913.
- (4) *Heart*, Lond., 1913-14.
- LEXER. (1) Die Aetiologie und die Mikroorganismen der akuten Osteomyelitis. *Samml. klin. Vortr.*, n. f. Leipz., 1897, No. 163.
- (2) Die entstehung entzündlicher Knochenherde und ihre Beziehungen zu den Arterienverzweigungen. *Arch. f. klin. Chir.*, Berl., 1903, lxxi.
- L'HEUREUX. Circulation artérielle auriculaire, ses rapports avec le faisceau de His, Lille, 1912, No. 33.
- LICHTHEIM. *Deutsch. Arch. f. klin. Med.*, Leipz., lxxx.
- LUKJANOW. Zur Lehre von den Funktionsstörungen einzelner Herzhöhlen, Vorläufige Mitteilung. *Centralbl. f. d. med. Wissensch.*, Berl., 1881, No. 49, and 1882, No. 18.

- LUSCHKA. (1) Das Endocardium und die Endocarditis. *Virchow's Arch.*, Berl., 1852.
- (2) Die Blutgefäße der Klappen des menschlichen Herzens. *Sitzungsb. d. k. k. Akad. d. Wissensch.*, Wien, math.-nat. Kl., 1859.
- (3) *Virchow's Arch.*, Berl., 1852.
- (4) Die Blutergüsse im Gewebe der Herzklappen. *Virchow's Arch.*, Berl., 1857.
- (5) Anatomie des Menschen, Tübingen, 1862, i, Part 2.
- MACCALLUM. A Textbook of Pathology, Philadelphia, 1916.
- MACKENZIE. Diseases of the Heart, London, 1910.
- MACNAMARA. Note on cardiac thrombosis. *Lancet*, 1879, xi.
- MCWILLIAM. Fibrillar contraction of the heart. *J. Physiol.*, Lond., 1887.
- MEIGS. The penetration of the muscle fiber of the human heart by capillaries and the existence in that organ of very large capillaries. *J. Anat. & Physiol.*, Lond., 1899.
- MERKEL, H. Zur Kenntniss der Kranzarterien des menschlichen Herzens. *Verhandl. d. deutsch. path. Gesellsch.*, Stuttg., 1906, x.
- MICHAELIS. Über einige Ergebnisse der Ligatur der Kranzarterien des Herzens. *Ztschr. f. klin. Med.*, Berl., 1897, xxiv.
- MILLER, W. S. A differential injection mass for use with stereoreöntgenograms. *Anat. Record*, 1918, xv.
- MILLER and MATTHEWS. Effect on the heart of experimental obstruction of the left coronary artery. *Arch. Int. Med.*, 1909, iii.
- MINELLI. Thrombosi ed embolia trombottica delle arterie coronarie del cuore. *Malad. d. cuore*, Roma, 1916-17, i.
- MOENCKEBERG. (1) Untersuchungen über Atrioventrikulärbündel im menschlichen Herzen, Jena, 1908.
- (2) Über die sogenannten abnormen Sehnenfäden im linken Ventrikel des menschlichen Herzens und ihre Beziehungen zum Atrioventrikulärbündel. *Verhandl. d. deutsch. path. Gesellsch.*, Berl.; Jena, 1908, xii.
- MORGAGNI. (1) De sedibus et causis morborum per anatomen indagatis, Venetiis, 1761, Lib. ii, xxiv, 16.
- (2) *Adversaria Anatomica*, Patavii, 1719.
- MÜLLER. Die Massenverhältnisse des menschlichen Herzens, Hamburg u. Leipzig, 1883.
- NUSSBAUM. (1) Über das Gefäßsystem des Herzens. *Arch. f. mikr. Anat.*, Bonn, 1912, lxxx.
- (2) *Frankfurt. Ztschr. f. Path.*, 1911, viii.
- ODINZOW. Vascularization der Herzklappen im Kindesalter. Inaug. Diss., München, 1904.
- OEHL. Sulla presenza dei elementi contrattili nella maggiore corda

- tendinea delle valvole mitrali umane. *Mem. dell' Acad. di Scienze di Torino*, 1861, xx.
- OERTEL. (1) The Anatomic-Histologic Processes of Bright's Disease, Philadelphia, 1910.
- (2) The physiologic degeneration of the mesonephros. *Arch. Int. Med.*, May, 1914, xiii.
- (3) On certain changes in the pancreas in diabetes mellitus, etc. *Scientific Reports, Royal Victoria Hospital*, Montreal, 1916, Series B, i.
- (4) Degeneration, senescence and new growth. *J. Med. Research*, July, 1918, 3, xxxviii.
- (5) The essential atrophy of the pancreas. *J. Med. Research*, September, 1919, xl, No. 3.
- (6) Postnatal development, etc. *Am. J. Med. Sc.*, May, 1921.
- OERTEL and ANDERSON. Regressive and progressive changes in the pancreas, etc. *Scientific Reports, Royal Victoria Hospital*, Montreal, 1916, Series B, i.
- OPITZ. Die Aktinogramme von einen Arteriosklerotiker und einem mit grauer Salbe injizierten Preparat. *Fortschr. a. d. Geb. d. Roentgenstrahlen*, Hamb., 1897, i.
- OPPENHEIMER and ROTHCHILD. Electrocardiographic changes associated with myocardial involvement with special reference to prognosis. *J. Am. M. Assn.*, 1917, lxix.
- OSLER. The Principles and Practice of Medicine, New York, 1905.
- PAGENSTECHER. Die Unterbindung der verletzten Arteria coronaria. *Deutsch. med. Wchnschr.*, Berl. and Leipz., 1901.
- PANUM. Experimentelle Beiträge zur Lehre von der Embolie. *Virchow's Arch.*, Berl., 1862, xxv.
- PARRY. An Inquiry into the Symptoms and Causes of the Syncope Anginosa, Commonly Called Angina Pectoris, London, 1799.
- PEACOCK. Rupture of heart, *Brit. M. J.* 1880, i.
- PICOT. Athérome des artères coronaires: thrombose de la coronaire antérieure; mort subite. *Sud-ouest méd.*, Bordeaux, 1880, i.
- PIQUAND. Recherches sur l'anatomie des vaisseaux sanguins du coeur. *J. de l'anat. et physiol.*, etc., Par. 1910, xlv.
- PORTER. (1) Über die Frage eines Coordinationcentrum im Herz-Ventrikel. *Arch. f. d. ges. Physiol.*, Bonn, 1893.
- (2) Results of ligation of the coronary artery. *J. Physiol.*, Lond., 1894, xv.
- PRATT. *Am. J. Physiol.*, vol. 1.
- PURKINJE. Mikroskopische neurologische Beobachtungen. *Arch. f. Anat. u. Physiol. (Müller's)*, Leipz., 1845.

- PUTJATIN. Über die pathologischen Veränderungen der automatischen Nervenganglien bei chronischen Herzkrankheiten. *Arch. f. path. Anat. u. Physiol.*, Berl., 1878, lxxiv.
- RECKLINGHAUSEN, VON and ZENKER. Über die Störungen des Myokardiums. *Verhandl. d. 10 int. med. Kong.*, Berlin, 1891.
- REDWITZ, VON. Der Einfluss der Erkrankungen der Koronararterien auf die Herzmuskulatur mit besonderer Berücksichtigung der chronischen Aortitis. *Virchow's Arch.*, Berl., 1909, excvii.
- REID. (1) On the measurements of the heart and tables on the weights of the most important organs of the body at different periods of life. *Heart*. Todd's Encyclopedia, London, 1843.
- REITMANN. Beiträge zu Pathologie der menschlichen Bauchspeicheldrüse. *Ztschr. f. Heilk.* (Abth. f. path. Anat.), Berl.; Wien, 1905, xxvi.
- RETZER. (1) Über die Muskulöse Verbindung zwischen Vorhof und ventrikel des Säugetierherzens. *Arch. f. Anat. u. Physiol.* (Anat. Abth.), Leipz., 1904, No. 1.
- (2) The atrioventricular bundle and Purkinje fibers. *Anat. Record*, 1906-8, i; also ii.
- (3) Anatomy of the conductive system in the mammalian heart. *Johns Hopkins Hosp. Bull.*, 1908, p. 208.
- RIBBERT. (1) *Virchow's Arch.*, Berl., cxlvii.
- (2) Lehrbuch der allgemeinen Pathologie und pathologischen Anatomie, Leipzig, 1908.
- RIOLANUS. *Anthropographia*, Lutetiae Parisiorum, 1649.
- ROKITANSKY. Handbuch der pathologischen Anatomie, Wien, 1842-46.
- ROPPE. Über Gefässe in den Herzklappen, Diss., Göttingen, 1904.
- ROSENOW. *J. Infect. Dis.*, 1912, xi.
- ROSENSTEIN. Handbuch der Krankheiten des Zirkulationsapparates, Ziemssen's Sammelwerk, Leipzig, 1876, vi.
- ROUGNON. Pathologisch-semiotische Betrachtungen aller Verrichtungen des menschlichen Körpers. Translated from the Latin, Leipzig, 1801, i.
- SAMUELSON. (1) Über den Einfluss der Koronararterienverschliessung auf die Herzaktion. *Ztschr. f. klin. Med.*, Berl., ii.
- (2) Über den Einfluss der Kranzarterienverschliessung auf die Herz-tätigkeit. *Centralbl. f. d. med. Wissensch.*, Berl. 1880, xviii.
- SAND, RENÉ. Les scléroses latentes et leur influence sur le pronostic des plaies de guerre. Contribution à l'étude du vieillissement pathologique. *Ambulance de l'océan. (La Panne)*, Paris, 1917, i. Fasc. 2.
- SAPPEY. *Traité d'Anatomie descriptive*, Paris, 1876.
- SCHULTHESS-RECHBERG. Über die Folgen der Kranzarterienverschliessung für das Herz. *Virchow's Arch.*, Berl., 1881, lxxxv.

- SÉE, BOCHEFONTAINE and ROUSSY. Arrêt rapide des contractions rythmiques, des ventricles cardiaques sous l'influence de l'occlusion des artères coronaires. *Comptes rendus*, Par., January 10, 1881.
- SÉNAC. Traité de la structure du coeur, Paris, 1774, 2d ed.
- SIDING, A. Über den Abschluss des Sinus coronarius cordis gegen den rechten Vorhof. *Anat. Anz.*, Jena, 1896.
- SKINNER, E. H. Circulatory opacity. *Am. Q. Roentgenol.*, 1912, iv.
- SMITH, F. M. (1) The ligation of coronary arteries with electrocardiographic study. *Arch. Int. Med.*, 1918, xxii.
- (2) Concerning the anatomy of the coronary arteries. *Am. J. M. Sc.*, 1918, clvi.
- SPALTEHOLZ. (1) Die Koronararterien des Herzens. *Verhandl. d. anat. Gesellsch.*, Würzburg, 1907, 21, Vers.
- (2) Zur vergleichender Anatomie der Arteriae coronariae cordis. *Verhandl. d. anat. Gesellsch.*, Berlin, 1908.
- (3) Über Arterien der Herzwand. *Verhandl. d. deutsch. path. Gesellsch.*, Leipzig, 1909.
- SPALTEHOLZ and HIRSCH. Koronarkreislauf und Herzmuskel, anatomische und experimentelle Untersuchungen. *Verhandl. d. 24 Kong. f. innere. Med.*, Wiesbaden, 1907, xxiv.
- STEGMANN, R. Ersatz des exstirpierten calcaneus durch Drehung des Talus. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, Hamb., 1905-6, ix.
- STENGEL. *Am. J. M. Sc.*, cxxx.
- STERNBERG. Über Erkrankungen des Herzmuskels im Anschluss an Störungen des Koronararterien Kreislaufes I. D., Marburg, 1887.
- TANDLER. Anatomie des Herzens. Bardeleben's Handbuch der Anatomie des Menschen, Jena, 1913, iii, Abt. 1.
- TAWARA. (1) Das Reitzleitungssystem des Säugetierherzens, Jena, 1906.
- (2) Über die sogenannten abnormen Sehnenfäden des Herzens. *Ziegler's Beitr.*, Jena, 1906, xxxix.
- TEDISCHI. Über die Fragmentation des Myocardium. *Virchow's Arch.*, Berl., 1892, cxxviii.
- THEBESIUS. Dissertatio de circulo sanguinis in corde, Lugdunum Batavorum, 1708.
- TIEILE. Traité myologie et d'angéiologie, Paris, 1843, xvi.
- THEILHABER. *München. med. Wchnschr.*, June 4, 1917.
- THOREL. (1) Vorläufige Mitteilungen über eine besondere Muskelverbindung zwischen der Cava superior und dem Hisschen Bündel. *München. med. Wchnschr.*, lvi, No. 42.
- (2) Ueber den Aufbau des Sinusknotens und seine Verbindung mit der Cava superior und den Wenckebachschen Bündeln. *München. med. Wchnschr.*, 1910, No. 4.

- TRENDELENBURG and COHN. Zur Physiologie des Übergangsbündels am Säugetierherzen. *Centralbl. f. Physiol.*, Leipz. u. Berl., 1909, xxiii.
- VALENTIN. Über die gegenseitigen Massenverhältnisse der rechten und der linken Kammer des Herzens. *Ztschr. f. rationelle Medizin*, Zürich, 1844, 1, 8.
- VERHEYEN. *Anatomiae corporis humani liber primus*, Coloniae, 1-12.
- VESALIUS. *De humani corporis fabrica libri septem*, 2d ed., Basileae, 1555.
- VIEUSSENS. (1) Nouvelle découvertes sur le cœur, Toulouse, 1706.
(2) *Traité nouveau de la structure et des causes du mouvement naturel du coeur*, Toulouse, 1715.
- WAUGH. Studies on the Bone-marrow in Disease, McGill Medical Society, Senior Prize Essay, 1920.
- WEED. *Johns Hopkins Hosp. Bull.*, October, 1920, xxxi.
- WENCKEBACH. (1) Beiträge zur Kenntniss der menschlichen Herztätigkeit. *Arch. f. Anat. u. Phys.* (Phys. Abteil.), Leipz., 1906 and 1907.
(2) *Arch. f. Physiol.*, Leipz., 1907, xx.
(3) *Arch. d. mal. du coeur etc.*, Par., 1908, No. 2.
- WHEELER. Slow pulse. *Brit. M. J.*, 1880, i.
- WILSON. Nerves of the atrioventricular bundle. *Anat. Record*, April, 1909.
- WINSLOW. *Exposition anatomique de la structure du corps humain*, Paris, 1776.
- WISSILIEWSKI. Zur Frage über den Einfluss der Embolie der Koronararterien auf die Herztätigkeit und den Blutdruck. *Ztschr. f. exper. Path. u. Therap.*, Berl., 1911, ix.
- WOOLLEY. Three cases which illustrate the consequences of coronary lesions. *J. Lab. & Clin. M.*, 1917, iii, 18.
- WYBAUW. Existe-t-il des vaisseaux sanguins dans les valvules du coeur? *Arch. d. mal. du coeur, etc.*, Par., 1918, xi.
- ZIMMERL. Ricerche anatomo-comparate sui vasi cardiaci degli animali domestici, Parma, 1900.

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